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MEDICO-CHIRURGICAL TRANSACTIONS,

PUBLISHED BY THE

ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

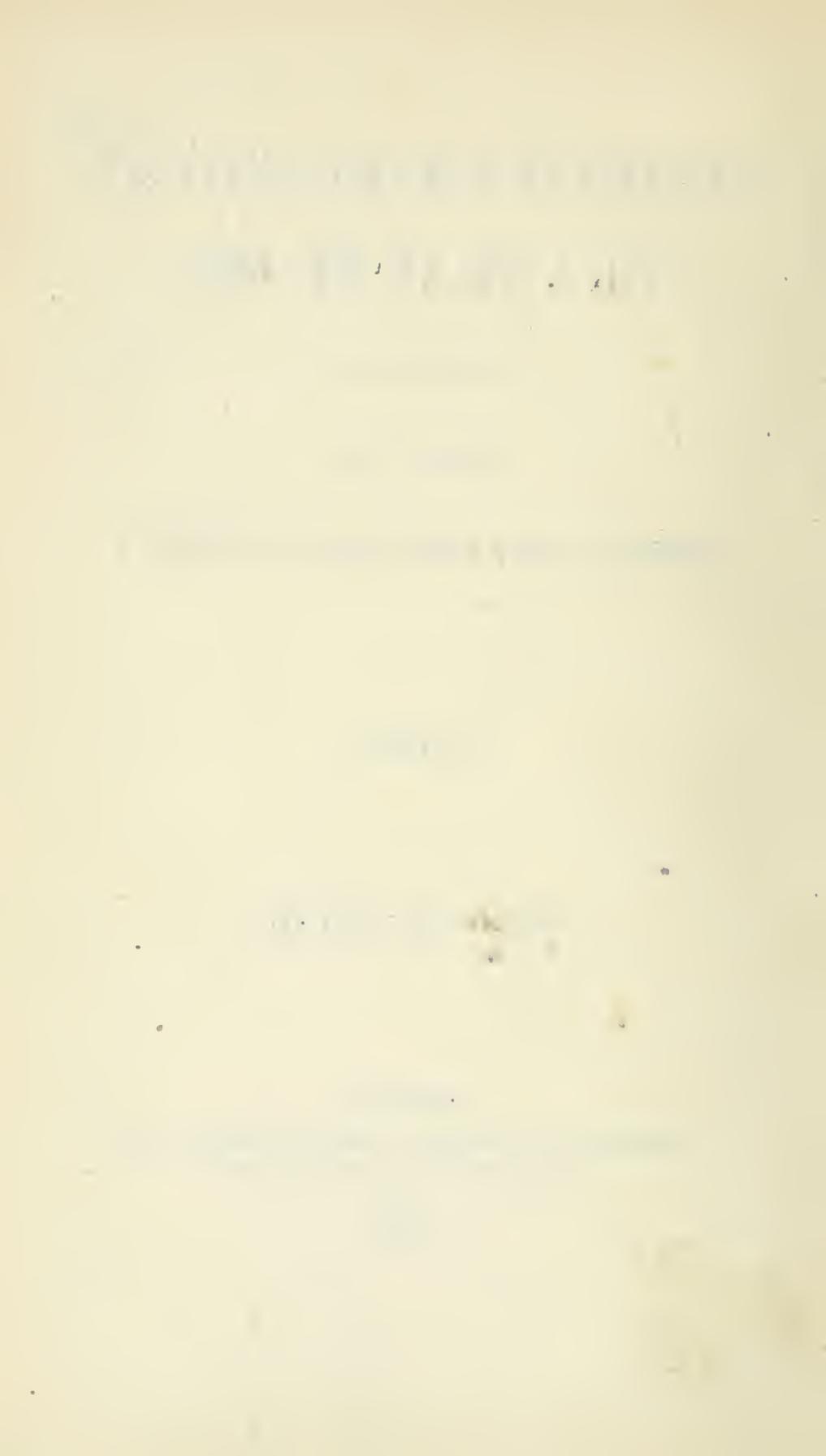
LONDON.

VOLUME THE THIRTIETH.

LONDON :

PRINTED FOR LONGMAN, BROWN, GREEN, AND
LONGMANS, PATERNOSTER-ROW.

1847.



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SECOND SERIES.

VOLUME THE TWELFTH.



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LONDON:
PRINTED BY RICHARD KINDER,
GREEN ARBOUR COURT, OLD BAILEY.

ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

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- 1805. WILLIAM SAUNDERS, M.D.
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- 1810. SIR HENRY HALFORD, BART., G.C.H.
- 1813. SIR GILBERT BLANE, BART.
- 1815. HENRY CLINE.
- 1817. WILLIAM BABINGTON, M.D.
- 1819. SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
- 1821. JOHN COOKE, M.D.
- 1823. JOHN ABERNETHY.
- 1825. GEORGE BIRKBECK, M.D.
- 1827. BENJAMIN TRAVERS.
- 1829. PETER MARK ROGET, M.D.
- 1831. WILLIAM LAWRENCE.
- 1833. JOHN ELLIOTSON, M.D.
- 1835. HENRY EARLE.
- 1837. RICHARD BRIGHT, M.D.
- 1839. SIR BENJAMIN COLLINS BRODIE, BART.
- 1841. ROBERT WILLIAMS, M.D.
- 1843. EDWARD STANLEY.
- 1845. WILLIAM F. CHAMBERS, M.D., K.C.H.
- 1847. JAMES MONCRIEFF ARNOTT, F.R.S.

F E L L O W S
OF THE
ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.

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L.—Librarian.	C.—Member of Council.

AUGUST 1847.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

ELECTED

1841 *JAMES ABERCROMBIE, M.D., *Cape of Good Hope.*
1846 John Abercrombie, M.D., *Physician to the Public Dispensary,
Lincoln's-inn; 13, Old Bond-street.*
1847 Elises Acosta, M.D., *Caraccas.*
1842 William Acton, *Surgeon to the Islington Dispensary; 46,
Queen Anne-street, Cavendish-square.*
1818 Walter Adam, M.D., *Physician to the Royal Public Dispensary,
Edinburgh.*
1818 Thomas Addison, M.D., *Physician to, and Lecturer on Medicine at,
Guy's Hospital; 24, New-street, Spring-gardens.
C. 1826. V. P. 1837.*
1814 Joseph Ager, M.D., *Great Portland-street. C. 1836.*
1819 *James Ainge, *Fareham, Hants.*
1837 *Ralph Fawsett Ainsworth, M.D., *104, King-street, Manchester.*
1819 George F. Albert.

ELECTED

1839 Rutherford Alcock, K.C.T., K.T.S., *China*.

1826 James Alderson, M.D., F.R.S., 36, *Charles-street, Berkeley-square*.

1843 C. J. B. Aldis, M.D., *Physician to the London and Surrey Dispensaries, and Lecturer on Medicine at the Charlotte-street School of Medicine*; 46 b, *Chester-square*.

1813 Henry Alexander, F.R.S., *Surgeon-Oculist in Ordinary to the Queen, and Surgeon to the Royal Infirmary for Diseases of the Eye*; 6, *Cork-street*. C. 1840.

1836 Henry Ancell, *Surgeon to the Western General Dispensary*; 3, *Norfolk-crescent, Oxford-square*. C. 1847.

1817 Alexander Anderson.

1816 John Goldwyer Andrews, *Surgeon to the London Hospital*; 4, *St. Helen's-place*. C. 1836. V.P. 1840.

1820 Thomas F. Andrews, M.D., *Norfolk, Virginia*.

1813 William Ankers, *Knutsford*.

1819 Professor Antommarchi, *Florence*.

1818 William Withering Arnold, M.D., *Physician to the Infirmary and Lunatic Asylum, Leicester*.

1825 Thomas Graham Arnold, M.D., *Stamford*.

1819 James M. Arnott, F.R.S., *PRESIDENT, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital*; *New Burlington-street*. L. 1826. T. 1835. V.P. 1832. C. 1846. P. 1847.

1828 Neil Arnott, M.D., F.R.S., *Physician Extraordinary to the Queen*; *Bedford-square*. C. 1835.

1817 John Ashburner, M.D., M.R.I.A., *Physician-Accoucheur to the Queen Charlotte's Lying-in Hospital*; 65, *Grosvenor-street*. C. 1821.

1841 John Avery, *Surgeon to the Charing Cross Hospital*; 3, *Queen-street, May Fair*.

1825 Benjamin Guy Babington, M.D., F.R.S., *Physician to Guy's Hospital, and Physician to the Deaf and Dumb Institution*; 31, *George-street, Hanover-square*. C. 1829. V.P. 1845.

1846 C. S. Metcalfe Babington, *Surgeon-Accoucheur to the Pimlico Dispensary*; 2, *Chester-street, Belgrave-square*.

1819 John Carr Badeley, M.D., *Chelmsford*.

1820 *John H. Badley, *Dudley*.

1838 Francis Badgley, M.D., *Montreal, Upper Canada*.

1840 William Bainbridge, *Upper Tooting*.

1836 Andrew Wood Baird, M.D., *Ipswich*.

ELECTED

1816 *William Baker, M.D., *Physician to the Derbyshire General Infirmary; Derby.*

1839 T: Graham Balfour, M.D., *Grenadier Guards, Army and Navy Club, St. James's-square.*

1837 William Baly, M.D., F.R.S., LIBRARIAN, *Physician to the General Penitentiary, Millbank, and Lecturer on Forensic Medicine at St. Bartholomew's Hospital; 28, Spring-gardens. C. 1845. L. 1847.*

1847 Andrew Whyte Barclay, M.D., 42, *Curzon-street, May Fair.*

1833 Thomas Alfred Barker, M.D., *Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Grosvenor-street. C. 1844.*

1843 Thomas Herbert Barker, *Priory-terrace, Bedford.*

1847 George Hilario Barlow, M.D., *Physician to Guy's Hospital; 7, Union-street, Southwark.*

1843 Christopher Hewetson Barnes, E.I.C.S.; *Belle-Vue House, Notting-hill.*

1815 *John Baron, M.D., *Cheltenham.*

1840 Benjamin Barrow, *Clifton.*

1844 William R. Basham, M.D., *Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Chester-street, Grosvenor-place.*

1836 William Beaumont, *Professor of Surgery in the University of King's College; Toronto, Upper Canada.*

1841 George Beaman, 32, *King-street, Covent-garden.*

1840 Charles Beevor, *Surgeon to the St. Marylebone Dispensary; 49, Berners-street.*

1818 *Joseph Bell, *Surgeon to the Royal Infirmary; Edinburgh.*

1819 Thomas Bell, F.R.S., L.S., and G.S., *Lecturer on Diseases of the Teeth, at Guy's Hospital; 17, New Broad-street. C. 1832.*

1847 Henry Bennet, M.D., *Cambridge-square, Hyde-park.*

1845 Edward Unwin Berry, *James-street, Covent-garden.*

1820 Stephen Bertin, *Paris.*

1818 John Jeremiah Bigsby, M.D., *Newark, Nottinghamshire.*

1827 William Birch, *Barton, Lichfield.*

1845 Golding Bird, M.D., F.R.S., *Assistant Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Myddelton-sq.*

1835 James Bird, 16, *Orchard-street, Portman-square.*

1846 Hugh Birt, *Morro Velhio, Minus Quæs, Rio Janeiro, Brazil; Surgeon to the Morro Velhio Hospital.*

ELECTED

1843 Patrick Black, M.D., *Assistant-Physician to St. Bartholomew's Hospital, and Physician to the Seamen's Hospital Ship "Dreadnought;" Bedford-square.*

1844 Thomas Blackall, M.D., *Physician to the Pimlico Dispensary; Queen-street, May-fair.*

1847 George C. Blackman, M.D., *New York, U.S.*

1839 Richard Blagden, *Surgeon-Accoucheur, and Surgeon Extraordinary, to the Queen; Surgeon in Ordinary to Her Royal Highness the Duchess of Kent; Albemarle-street. C. 1847.*

1814 Thomas Blair, M.D., *Physician to the Sussex County Hospital; Brighton.*

1841 James Blake.

1840 Peyton Blakiston, M.D., F.R.S., *Birmingham.*

1845 Henry Blenkinsop, *Warwick.*

1811 *Henry C. Boisragon, M.D., *Cheltenham.*

1823 Louis Henry Bojanus, M.D., *Wilna.*

1816 Hugh Bone, M.D., *Inspector-General of Hospitals.*

1810 John Booth, M.D., *Physician to the General Hospital at Birmingham.*

1846 John Ashton Bostock, *Scots Fusileer Guards.*

1846 Peter Bossey, *Surgeon to the Hulks at Woolwich; Thomas-street, Woolwich.*

1841 William Bowman, F.R.S., *Assistant-Surgeon to King's College Hospital, and to the Royal Ophthalmic Hospital, Moorfields; 14, Golden-square.*

1844 Robert Brandon.

1814 Richard Bright, M.D., F.R.S., *Physician Extraordinary to the Queen, and Consulting Physician to Guy's Hospital; Saville-row. C. 1821. V. P. 1827. P. 1837.*

1813 Sir Benjamin Collins Brodie, Bart., F.R.S., *Sergeant-Surgeon to the Queen, Surgeon in Ordinary to His Royal Highness Prince Albert, Foreign Correspondent of the Institute of France, and Foreign Associate of the Royal Academy of Medicine of Paris; Saville-row. C. 1814. V. P. 1816. P. 1839.*

1844 Charles Brooke, F.R.S., *Keppel-street, Russell-square.*

1847 George Brown, *Grenadier Guards Hospital; Rochester-row, Westminster.*

1847 *Robert Brown, *Winckley-square, Preston, Lancashire.*

ELECTED

1842 Charles Blakely Brown, M.B., *Physician-Accoucheur to the St. George's and St. James's Dispensary*; 38, Hill-street, Berkeley-square

1818 *Samuel Barwick Bruce, *Surgeon to the Forces*; Ripon.

M. Pierre Brulatour, *Surgeon to the Hospital*; Bourdeaux.

1823 B. Bartlet Buchanan, M.D.

1843 J. Charles Bucknill, M.B., *Exminster, Devonshire.*

1839 George Budd, M.D., F.R.S., *Fellow of Caius College, Cambridge*; *Professor of Medicine in King's College, London*; *Physician to King's College Hospital*; 20, Dover-street, Piccadilly. C. 1846.

1839 Thomas Henry Burgess, M.D.

1844 A. J. Burmester, M.D., *Malta.*

1833 George Burrows, M.D., F.R.S., *TREASURER*, *Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital*; 45, Queen Anne-street. C. 1839. T. 1845.

1820 Samuel Burrows.

1835 Henry Burton, M.D., *Physician to St. Thomas's Hospital*; 41, Jermyn-street. C. 1842.

1837 George Busk, *Surgeon to the Hospital-ship Dreadnought*; Croom's-hill, Greenwich. C. 1847.

1818 John Butter, M.D., F.R.S., F.L.S., *Physician to the Plymouth Eye Infirmary*; Plymouth.

1832 *William Campbell, M.D., *Physician to the New Town Dispensary, and Lecturer on Midwifery*; Edinburgh.

1842 Henry Cantis, 8, Maddox-street, Hanover-square.

1846 John Burford Carlill, 57, Berners-street.

1839 Robert Carswell, M.D., *Physician to their Majesties the King and Queen of the Belgians*; Brussels.

1825 Harry Carter, M.D., *Physician to the Kent and Canterbury Hospital*; Canterbury.

1818 Richard Cartwright, 34, Bloomsbury-square.

1820 Samuel Cartwright, F.R.S., Nizell's-house, near Tonbridge.

1845 Samuel Cartwright, Jun., Sackville-street, Piccadilly.

1839 William Cathrow, Weymouth-street.

1845 William Oliver Chalk, Nottingham-terrace, New-road.

1818 Richard Chamberlaine, Kingston, Jumaiica.

1816 William Frederick Chambers, K.C.H., M.D., F.R.S., *Physician to the Queen, and to the Queen Dowager*: 46, Brook-street. C. 1818. V. P. 1821. P. 1845.

ELECTED

1844 Thomas King Chambers, M.D., 1, *Hill-street, Berkeley-square.*

1837 Henry T. Chapman.

1838 George Chaplin Child, M.D., *Physician to the Westminster General Dispensary; 27, Mortimer-street.*

1842 W. D. Chowne, M.D., *Physician to the Charing Cross Hospital; 8, Connaught-place West, Hyde-park.*

1827 Sir James Clark, Bart., M.D., F.R.S., *Physician to the Queen, Physician in Ordinary to His Royal Highness Prince Albert, and Consulting Physician to their Majesties the King and Queen of the Belgians; Brook-street. C. 1830. V. P. 1832.*

1839 Frederick Le Gros Clark, SECRETARY, *Assistant-Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, St. Thomas's Hospital; Finsbury-place. S. 1847.*

1845 John Clark, M.D., *Staff Surgeon 2nd class; 12, Beaumont-street, Portland-place.*

1847 Benjamin Clark, *Brook-street, Grosvenor-square.*

1835 James Clayton, 3, *Percy-street, Bedford-square.*

1842 Oscar M. P. Clayton, 3, *Percy-street, Bedford-square.*

1827 John Clendinning, M.D., F.R.S., *Physician to the St Marylebone Infirmary; 16, Wimpole-street. C. 1832. S. 1834. V. P. 1840.*

1835 *William Colborne, *Chippingham, Wilts.*

1818 Robert Cole, F.L.S., *Holybourne, Hampshire.*

1847 James Milman Coley, M.D., 47, *Chester-square.*

1828 John Conolly, M.D., *Hanwell.*

1839 John C. Cooke, M.D., *Caroline-street, Bedford-square.*

1840 *William Robert Cooke, *Burford, Oxfordshire.*

1840 Bransby Blake Cooper, F.R.S., *Surgeon to, and Lecturer on Surgery at, Guy's Hospital; New-street, Spring-gardens. C. 1830. V. P. 1842.*

1820 Benjamin Cooper, *Stamford.*

1819 George Cooper, *Brentford.*

1841 George Lewis Cooper, *Surgeon to the Bloomsbury Dispensary; 35, Keppel-street, Russell-square.*

1843 William White Cooper, *Senior Surgeon to the North London Ophthalmic Institution, and to the Honourable Artillery Company; 2, Tenterden-street, Hanover-square.*

ELECTED

1841 Holmes Coote, *Demonstrator of Anatomy at St. Bartholomew's Hospital, Surgeon to the North London Ophthalmic Institution; 14, Southampton-street, Bloomsbury.*

1835 George F. Copeland, *Cheltenham.*

1822 James Copland, M.D., F.R.S., *Consulting Physician to Queen Charlotte's Lying-in Hospital; 5, Old Burlington-street. C. 1830. V. P. 1838.*

1847 John Rose Cormack, M.D., *Putney, Surrey.*

1839 *Charles C. Corsellis, M.D., *Resident Physician to the Lunatic Asylum, Wakefield.*

1814 *William Cother, *Surgeon to the Infirmary, Gloucester.*

1828 William Coulson, *Surgeon to the Magdalen Hospital, Consulting Surgeon to the City Lying-in Hospital; Frederick's-place, Old Jewry. C. 1831. L. 1832.*

1836 William Travers Cox, M.D., 2, *Stanhope-place, Hyde-park.*

1817 Sir Philip Crampton, Bart., F.R.S., *Surgeon-General to the Forces in Ireland.*

1814 Stewart Crawford, M.D., *Bath.*

1841 M. A. N. Crawford, M.D., *Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 62, Upper Berkeley-street, Portman-square.*

1822 Sir Alexander Crichton, M.D., F.R.S., and F.L.S., *Physician in Ordinary to their Imperial Majesties the Emperor and Dowager Empress of all the Russias. C. 1823.*

1847 George Critchett, *Assistant-Surgeon to the London Hospital, and the Royal London Ophthalmic Hospital; Broad-street-buildings.*

1837 John Farrar Crookes, 21, *Argyll-street.*

1820 John Green Crosse, M.D., F.R.S., *Surgeon to the Norfolk and Norwich Hospital.*

1812 *Henchman Crowfoot, *Beccles.*

1818 William Cuming, M.D., *Professor of Botany at the Glasgow Institution, and Surgeon to the Royal Infirmary at Glasgow.*

1837 Thomas Blizard Curling, *Lecturer on Surgery at, and Assistant-Surgeon to, the London Hospital; 37, New Broad-street. S. 1845.*

1846 Henry Curling, *Ramsgate.*

1847 J. Edmund Currey, 16, *Pall-mall.*

1836 George Cursham, M.D., *SECRETARY, Physician to the Asylum for Female Orphans; 5, Saville-row. S. 1842.*

ELECTED

1847 Henry Charles Curtis, 45, *Davies-street, Grosvenor-square.*
 1822 Christopher John Cusack.
 1828 Adolphe Dalmas, M.D., *Paris.*
 1840 John Dalrymple, *Surgeon to the London Ophthalmic Hospital;*
 56, *Grosvenor-street.*
 1836 *James S. Daniel, *Ramsgate.*
 1820 George Darling, M.D., 6, *Russell-square.* C. 1841.
 1818 *Sir Francis Sacheverel Darwin, Knt., M.D., *Sydone, near*
 Matlock, Derbyshire.
 1818 Henry Davies, M.D., *Physician to the British Lying-in*
 Hospital, Brownlow-street; Physician-Accoucheur to the
 Marylebone General Dispensary; Saville-row. C. 1827.
 1846 Frederick Davies, *Upper Gower-street, Bedford-square.*
 1847 John Davies, M.D., *Physician to the Hertford Infirmary, and*
 visiting Physician to the County Goal and Lunatic Asy-
 lum, Hertford.
 1820 Thomas Davis, *Brook-street, Hanover-square.* C. 1843.
 1818 James Dawson, *Liverpool.*
 1841 Campbell De Morgan, *Assistant-Surgeon to, and Lecturer on*
 Anatomy at, the Middlesex Hospital; 17, Manchester-
 street.
 1846 *Samuel Best Denton, *Ivy-lodge, Hornsea, East Riding,*
 Yorkshire.
 1816 *Sir David James Hamilton Dickson, M.D., F.R.S. Ed., and
 F.L.S., *Physician to the Fleet, and to the Royal Naval*
 Hospital, Plymouth.
 1839 James Dixon, *Surgeon to the Royal London Ophthalmic*
 Hospital, 37, Broad-street-buildings.
 1844 Robert Dickson, M.D., *Curzon-street, May Fair.*
 1845 John Dodd, *Portman-street, Portman-square.*
 1826 John Sommers Down, M.D., *Southampton.*
 1839 Henry Pye Lewis Drew, 79, *Gower-street, Bedford-square.*
 1846 John Drummond, *Deputy Inspector of Fleets and Hospitals;*
 Royal Naval Hospital, Woolwich.
 1843 Thomas Jones Drury, M.D., *Physician to the Salop Infirmary;*
 Shrewsbury.
 1845 George Duff, M.D., *Genoa.*
 1845 Edward Duffin, *Langham-place, Portland-place.*
 1833 William Dunbar, M.D., *Bombay.*
 1833 Robert Dunn, *Norfolk-street, Strand.* C. 1845.

ELECTED

1843 C. M. Durrant, M.D., *Physician to the East Suffolk and Ipswich Hospital; Ipswich.*

1839 Henry Dyer, M.D., 37, *Bryanston-square.*

1836 J. W. Earle, *Cheltenham.*

1824 George Edwards.

1823 C. C. Egerton, *India.*

1814 Philip Elliot, M.D., *Bath.*

1838 Thomas Elliotson, M.D., *Clapham.*

1835 William England, M.D., *Wisbeach.*

1842 John E. Erichsen, *Lecturer on General Anatomy and Physiology at the Westminster Hospital; 48, Welbeck-street, Cavendish-square.*

1815 G. F. D. Evans, M.D., *Hill-street, Berkeley-square.* C. 1838.

1836 George F. Evans, M.B., *Physician to the Birmingham Hospital.*

1845 William Julian Evans, M.D.

1841 Sir James Eyre, M.D., *Physician-Accoucheur to St. George's and St. James's Dispensary; 11, Brook-street, Grosvenor-square.*

1844 Arthur Farre, M.D., F.R.S., *Professor of Midwifery in King's College, London; Curzon-street, May Fair.*

1831 Robert Ferguson, M.D., *VICE-PRESIDENT, Physician-Accoucheur to the Queen, Physician to the Westminster Lying-in Hospital; Queen-street, May Fair.* C. 1839. V. P. 1847.

1841 William Fergusson, F.R.S. Ed., *Professor of Surgery in King's College, London; and Surgeon to King's College Hospital; 8, Dover-street, Piccadilly.*

1841 William Finch, M.D., F.L.S., *Laverstock-hall, Salisbury.*

1836 John W. Fisher, *Surgeon-in-Chief to the Metropolitan Police Force; 7, Upper Grosvenor-street.* C. 1843.

1839 George Lionel Fitzmaurice, 97, *Gloucester-place, Portman-square.*

1842 Thomas Bell Elecock Fletcher, M.D., *Physician to the General Dispensary, Birmingham.*

1841 John Forbes, M.D., F.R.S., *Physician to Her Majesty's Household; Old Burlington-street.*

1817 *Robert T. Forster, *Southwell.*

1820 Thomas Forster, M.D., *Hartfield-lodge, East Grinstead.*

ELECTED

1846 Algernon Frampton, M.D., *Physician to the London Hospital; New Broad-street.*

1816 John W. Francis, M.D., *Professor of Materia Medica in the University of New York.*

1841 J. Ch. August. Franz, M.D., *Royal German Spa, Brighton.*

1843 Patrick Fraser, M.D., *Assistant-Physician to the London Hospital; Guildford-street, Russell-square.*

1846 Joseph Freeman, *Spring-gardens.*

1836 John G. French, *Surgeon to St. James's Infirmary; 41, Great Marlborough-street.*

1846 Henry William Fuller, M.B., 45, *Half Moon-street Piccadilly.*

1815 *George Frederick Furnival, *Egham.*

1819 John Samuel Gaskoin, 32, *Clarges-street.* C. 1836.

1819 Henry Gaulter.

1821 *Richard Francis George, *Surgeon to the Bath Hospital.*

1841 J. Durancè George, F.G.S., *Lecturer on Dental Surgery at University College, and Dental Surgeon to University College Hospital; 32, Old Burlington-street.*

1812 George Goldie, M.D., *York.*

1837 Richard H. Goolden, M.D., *Assistant-Physician to St. Thomas's Hospital; 41, Sussex-gardens, Hyde-park.*

1818 James Alexander Gordon, M.D., F.R.S. C. 1828. V.P. 1829.

1844 John Grantham, *Crayford, Kent.*

1846 George Thompson Gream, *Surgeon to Queen Charlotte's Lying-in Hospital; 42, Hertford-street, May Fair.*

1816 Joseph Henry Green, F.R.S., *Surgeon to St. Thomas's Hospital; Hadley, Middlesex.* C. 1820. V. P. 1830.

1841 George Gregory, M.D., *Physician to the Small-pox Hospital; 31, Weymouth-street.* S. 1825.

1843 Robert Greenhalgh, *Upper Charlotte-street, Fitzroy-square.*

1835 William Griffith, *Surgeon to the Royal Maternity Charity; Lower Belgrave-street, Belgrave-square.*

1814 John Grove, M.D., *Salisbury.*

1837 James Manby Gully, M.D., *Holyrood-house, Great Malvern.*

1819 John Gunning, *Inspector of Hospitals; Paris.*

1841 Charles Guthrie, *Assistant-Surgeon to the Westminster Ophthalmic Hospital; Saville-row.*

1827 Marshall Hall, M.D., F.R.S., *Manchester-square.* L. 1828. C. 1840. V. P. 1843.

ELECTED

1842 *George Hall, M.D., 14, *Old Steine, Brighton.*

1845 John Hall, M.D., *Deputy Inspector-General of Hospitals; Cape of Good Hope.*

1819 Thomas Hammerton, 111, *Piccadilly.* C. 1829.

1838 Henry Haneock, *Surgeon to the Charing Cross Hospital; Harley-street.*

1836 J. F. Harding, 6, *Mylne-street, Myddelton-square.*

1843 Thomas Sunderland Harrison, M.D., F.L.S., *Garston-lodge, Somersetshire.*

1846 John Harrison, *The Court-yard, Albany.*

1841 William Harvey, *Surgeon to the Freemasons' Female Charity; Soho-square.*

1845 John Havers, *Bedford-place, Russell-square.*

1816 *John Haviland, M.D., *Regius Professor of Physic in the University of Cambridge; Physician to Addenbroke's Hospital; Cambridge.*

1828 Cæsar Hawkins, *Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 26, Lower Grosvenor-street.* C. 1830. V. P. 1838. T. 1841.

1838 Charles Hawkins, *The Court-yard, Albany.* C. 1846.

1820 Thomas Emerson Headlam, M.D., *Newcastle-upon-Tyne.*

1829 Thomas Heberden, M.D., 11, *Upper Brook-street.*

1844 John Hennen, M.D., *Physician to the Western General Dispensary; 24, Upper Southwick-street, Hyde-park.*

1821 Vincent Herberski, M.D., *Professor of Medicine in the University of Wilna.*

1843 Prescott Gardner Hewett, *Lecturer on Anatomy at St. George's Hospital Medical School; Vigo-street, Burlington-gardens.*

1841 *Nathaniel Highmore, *Consulting-Surgeon to the Weymouth and Dorsetshire Eye Infirmary; Sherborne.*

1814 *William Hill, *Wooton-under-Edge.*

1842 William Augustus Hillman, *Surgeon to the Furringdon Dispensary; Argyll-street.*

1841 John Hilton, F.R.S., *Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 10, New Broad-street.*

1840 Thomas Hodgkin, M.D., 9, *Brook-street.* C. 1842.

1813 Joseph Hodgson, F.R.S., *Surgeon to the General Hospital, and to the Eye Infirmary, Birmingham.* C. 1817.

1835 T. H. Holberton, *Surgeon Extraordinary to the Queen Dowager; Hampton.*

ELECTED

1843 Luther Holden, 39, *Ely-place, Holborn.*

1814 Henry Holland, M.D., F.R.S., *Physician Extraordinary to the Queen, and Physician in Ordinary to His Royal Highness Prince Albert; 25, Brook-street. C. 1817. V. P. 1826.*

1846 Bernard William Holt, *Assistant-Surgeon to the Westminster Hospital; Abingdon-street, Westminster.*

1846 Carsten Holthouse, *Surgeon to the Public Dispensary, Lincoln's Inn; Lecturer on Anatomy and Physiology; 3, Serle-street, Lincoln's-inn-fields.*

1819 *John Howell, M.D., F.R.S. Ed.; *Clifton.*

1828 *Edward Howell, M.D., *Swansea.*

1844 Edwin Humby, *Windsor-terrace, Maida-hill.*

1822 Robert Hume, M.D., *Inspector of Hospitals; Commissioner in Lunacy; 9, Curzon-street. V. P. 1836.*

1840 Henry Hunt, M.D., *Brook-street, Hanover-square.*

1842 Christopher Hunter, *Downham, Norfolk.*

1820 William Hutchinson, M.D.

1840 Charles Hutton, M.D., *Physician to the Royal Infirmary for Children; 25, Motcomb-street, Belgrave-square.*

1838 William Igl, M.D.

1847 William Edmund Image, *Surgeon to the Suffolk General Hospital; Bury St. Edmund's.*

1826 William Ingram, *Midhurst.*

1839 A. R. Jackson, M.D., *East India Company's Depôt, Warley Barracks, Essex.*

1845 *Henry Jackson, *Surgeon to the Sheffield General Infirmary; St. James's Row, Sheffield.*

1841 Paul Jackson, *Thayer-street, Manchester-square.*

1847 Thomas Reynolds Jackson, 28, *Charles-street, St. James's.*

1841 Maximilian M. Jacobovicz, M.D., *Pesth.*

1825 John B. James, M.D.

1847 William Withall James, *Exeter, Devon.*

1844 Samuel John Jeafferson, M.D., *Warwick.*

1839 Julius Jeffreys, F.R.S., 25 λ , *Norfolk-crescent, Hyde-park.*

1840 *G. Samuel Jenks, M.D., *Brighton.*

1821 Edward Johnson, M.D., *Weymouth.*

1837 Henry Charles Johnson, *Assistant-Surgeon to, and Lecturer on Medical Jurisprudence at, St. George's Hospital Saville-row.*

ELECTED

1844 John Johnston, 16, *Dover-street, Piccadilly.*

1844 Henry Bence Jones, M.A., F.R.S., *Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; Lower Grosvenor-street.*

1835 H. T. Jones, 20, *Soho-square.*

1837 T. W. Jones, M.D., 19, *Finsbury-pavement, Finsbury-square.*

1829 *G. Julius, *Richmond.*

1816 *George Hermann Kauffmann, M.D., *Hanover.*

1815 Robert Keate, *Sergeant-Surgeon to the Queen, Surgeon to the Queen Dowager, to H.R.H. the Duke of Cambridge, to H.R.H. the Duchess of Gloucester, and to St. George's Hospital; Albemarle-street.* C. 1818. V. P. 1826.

1838 Lionel P. Kell, *Bridge-street, Westminster.*

1847 Alfred Keyser, *Norfolk-crescent, Oxford-square.*

1839 *David King, M.D., *Eltham.*

1836 P. Nugent Kingston, M.D., *Physician to, and Lecturer on Medicine at, the Westminster Hospital; Curzon-street, May Fair.* C. 1846.

1806 James Laird, M.D.

1840 Samuel Lane, *Assistant-Surgeon to the Lock Hospital, and Lecturer on Anatomy; Grosvenor-place.*

1841 *Charles Lashmar, M.D., *Croydon, Surrey.*

1816 G. E. Lawrence.

1809 William Lawrence, F.R.S., *Surgeon Extraordinary to the Queen; Surgeon to St. Bartholomew's Hospital, and to Bridewell and Bethlem Hospitals; Lecturer on Surgery at St. Bartholomew's Hospital; 18, Whitehall-place.* S. 1813. V. P. 1818. C. 1820. T. 1821. P. 1831.

1840 Thomas Laycock, M.D., *York.*
*Jesse Leach, *Heywood, near Bury, Lancashire.*

1823 John G. Leath, M.D.

1822 John Joseph Ledsam, *Surgeon to the Birmingham Eye Infirmary.*

1822 Robert Lee, M.D., F.R.S., *Physician to the British Lying-in Hospital, and Physician-Accoucheur to the St. Mary-lebone Infirmary; Lecturer on Midwifery at St. George's Hospital; 4, Saville-row.* C. 1829. S. 1830. V. P. 1835.

1823 Henry Lee, M.D., 20, *Keppel-street, Russell-square.* C. 1837.
S. 1839.

1842 Edwin Lee, *Curzon-street, May Fair.*

ELECTED

1843 Henry Lee, *Lecturer on Physiology at St. George's Hospital Medical-school; 3, Dover-street, Piccadilly.*

1846 Thomas Sufford Lee, 2, *Upper Gordon-street, Euston-square.*

1843 John Leeson, 4, *Finsbury-square.*

1836 Frederick Leighton, M.D.

1847 John C. W. Lever, M.D., *Wellington-street, Borough.*

1847 John Liddle, M.D., *Inspector of Hospitals; Royal Hospital, Greenwich.*

1806 John Lind, M.D.

1835 Robert Liston, F.R.S., *Vice-PRESIDENT, Surgeon to University College Hospital; 5, Clifford-street, Bond-street.* C. 1840. V. P. 1846.

1845 William Little, M.D., *Physician to, and Lecturer on Medicine at, the London Hospital; Finsbury-square.*

1818 Robert Lloyd, M.D.

1824 Eusebius Arthur Lloyd, *Surgeon to St. Bartholomew's Hospital, and Surgeon to Christ's Hospital; 14, Bedford-row.* S. 1827. V. P. 1838. C. 1843.

1820 J. G. Locher, M.C.D., *Town Physician of Zurich.*

1824 Charles Locock, M.D., *First Physician-Accoucheur to the Queen; Physician to the Queen Dowager, and to the Westminster Lying-in Hospital; Hertford-street, May Fair.* C. 1826. V. P. 1841.

1846 Henry Thomas Lomax, *Stafford.*

1844 Edward Lonsdale, *Assistant-Surgeon to the Orthopædic Institution; Guildford-street, Russell-square.*

1836 Joseph S. Löwenfeld, M.D., *Berbice.*

1815 *Peter Luard, M.D.

1846 William Salmon Lucas, 2, *Dowgate-hill, City.*

1814 Sir James Macgrigor, Bart., K.T.S., M.D., LL.D., F.R.S. L. and Ed. *Director-General of the Medical Department of the Army; 3, Harley-street, Cavendish-square.* C. 1820. V. P. 1815.

1823 George Macilwain, *Consulting Surgeon to the Finsbury Dispensary; The Court-yard, Albany.* C. 1829.

1818 W. Mackenzie, *Surgeon to the Eye Infirmary, Glasgow.*

1822 Richard Mackintosh, M.D.

1847 Henry John McDougall, 46, *Berners-street.*

1846 William M'Ewen, M.D., *Surgeon to the Cheshire County Gaol, and House-Surgeon to the Chester General Infirmary; Newgate-street, Chester.*

ELECTED

1839 William Macintyre, M.D., *Harley-street.*

1844 Daniel MacLachlan, M.D., *Physician to the Royal Hospital, Chelsea.*

1842 John Macnaught, M.D., *Bedford-street, Liverpool.*

1835 D. C. Macreight, M.D., *St. Hillier's, Jersey.*

1837 A. M. M'Whinnie, *Demonstrator of Anatomy, and Lecturer on Comparative Anatomy, at St. Bartholomew's Hospital; Bridge-street, Blackfriars.*

1836 John Malyn, *Surgeon to the Western Dispensary, and to the Infirmary of St. Margaret and St. John; 12, James-street, Buckingham-gate.*

1824 Sir Henry Marsh, Bart., M.D., *Dublin.*

1838 Thomas Parr Marsh, M.D., *Physician to the Salop Infirmary, Shrewsbury.*

1840 John Marston, 6, *Devonshire-street, Portland-place.*

1841 James Ranald Martin, F.R.S., 71 A, *Grosvenor-street.*

1819 *John Masfen, *Surgeon to the County General Infirmary, and Fever Hospital, Stafford.*

1818 J. P. Maunoir, *Professor of Surgery at Geneva.*

1820 Herbert Mayo, F.R.S. S. 1825. V. P. 1834.

1837 Thomas Mayo, M.D., F.R.S., *Physician to the St. Marylebone Infirmary; Wimpole-street. S. 1841. C. 1847.*

1839 R. H. Meade, *Bradford, Yorkshire.*

1819 *Thomas Medhurst, *Hurstbourne Tarrant.*

1847 Edward Meryon, M.D., 14, *Clarges-street, Piccadilly.*

1811 Samuel Merriman, M.D., F.L.S., 34, *Brook-street. C. 1813. V. P. 1827. T. 1837.*

1837 S. W. J. Merriman, M.D., *Physician to the Westminster General Dispensary; Brook-street.*

1815 Augustus Meyer, M.D., *St. Petersburgh.*

1840 Richard Middlemore, *Surgeon to the Eye Infirmary, Birmingham.*

1818 *Patrick Miller, M.D., F.R.S. Ed., *Physician to the Devon and Exeter Hospitals, and to the Lunatic Asylum; Exeter.*

1844 Nathaniel Montefiore.

1828 Joseph Moore, M.D., *Physician to the Royal Freemasons' Female Charity; 10, Saville-row. C. 1837.*

1836 George Moore, M.D., *Hastings.*

1842 Thomas Morton, *Assistant-Surgeon to University College Hospital, and Surgeon to the Queen's Prison; 7, Woburn-place, Russell-square.*

ELECTED

1847 Simon Murchison, 7, Grosvenor-street.

1814 *George Frederick Muhr, M.D., Hanover.

1841 Edward William Murphy, M.D., *Professor of Midwifery in University College; Henrietta-street, Cavendish-square.*

1845 Thomas D. Mütter, M.D., *Professor of Surgery in Jefferson Medical College; Philadelphia.*

1840 Robert Nairne, M.D., *Physician to, and Lecturer on Medicine at, St. George's Hospital; 44, Charles-street, Berkeley-square.*

1805 Thomas Nelson, M.D., *Tonbridge Wells.* C. 1810. V.P. 1836.

1835 Thomas Andrew Nelson, M.D., 41, George-street, Portman-square.

1843 Edward Newton, *Howland-street, Fitzroy-square.*

1816 Thomas Nixon (Army).

1819 *George Norman, *Surgeon to the United Hospital and Puerperal Charity; Bath.*

1845 Henry Norris, *South Petherton, Somerset.*

1829 John North, *Gloucester-place.* C. 1835.

1843 William O'Connor, 21, George-street, Portman-square.

1847 Thomas O'Connor, *March, Cambridgeshire.*

1846 Francis Odling, *Devonshire-street, Portland-place.*

1822 James Adey Ogle, M.D., F.R.S., *Clinical and Aldrichian Professor of Medicine, Oxford; and Senior Physician to the Radcliffe Infirmary.*

1842 William Piers Ormerod, *Surgeon to the Radcliffe Infirmary, Oxford.*

1846 Edward Latham Ormerod, M.B., *Demonstrator of Morbid Anatomy at St. Bartholomew's Hospital; 10, Somerset-street, Portman-square.*

1844 Drewry Ottley, *Bedford-place, Russell-square.*

1847 William Emanuel Page, M.D., *Physician to St. George's Hospital; Curzon-street, May Fair.*

1847 *William Bonsfield Page, *Surgeon to the Cumberland Infirmary; Carlisle.*

1840 James Paget, *Assistant-Surgeon to, and Lecturer on General and Morbid Anatomy and Physiology, and Warden of the Collegiate Establishment at, St. Bartholomew's Hospital.*

1806 *Robert Paley, M.D., *Bishopston-grange, near Ripon, Yorkshire.*

ELECTED

1814 John Ranicar Park, M.D.

1836 J. W. Langston Parker, *Birmingham.*

1843 *Charles Lewis Parker, A.M., *Surgeon to the Radcliffe Infirmary, Oxford.*

1847 Nicholas Parker, M.B., *Microscopical Demonstrator of Morbid Anatomy at the London Hospital School of Medicine; Finsbury-square.*

1841 John Parkin, 157, *Sloane-street.*

1828 Richard Partridge, F.R.S., *VICE-PRESIDENT, Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New-street, Spring-gardens.* S. 1832. C. 1837. V. P. 1847.

1819 Granville Sharp Pattison, *New York.*

1845 Thomas Bevill Peacock, M.D., *Physician to the Royal Free Hospital and Royal General Dispensary; 20, Finsbury-circus.*

1830 Charles P. Pelechin, M.D., *St. Petersburgh.*

1830 William Pennington, 21, *Montague-place, Russell-square.*

1819 John Pryor Peregrine, *Penzance, Cornwall.*

1839 Thomas Peregrine, 3, *Half-moon-street.*

1831 Jonathan Pereira, M.D., F.R.S., F.L.S., *VICE-PRESIDENT, Assistant-Physician to, and Lecturer on Materia Medica at, the London Hospital; Finsbury-square.* C. 1844. V. P. 1847.

1836 John George Perry, F.G.S., *Inspector of Prisons; 12, Westbourne-street, Hyde-park-gardens.* C. 1831. S. 1837. V. P. 1845.

1844 William V. Pettigrew, 30, *Chester-street, Grosvenor-place.*

1814 *Edward Phillips, M.D., *Physician to the County Hospital; Winchester.*

1837 Benjamin Phillips, F.R.S., *TREASURER, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 17, Wimpole-street.* L. 1841. T. 1847.

1846 Francis Richard Philp, M.D., *Physician to St. Luke's Hospital; 28, Grosvenor-street.*

1836 Isaac Pidduck, M.D., 22, *Montague-street, Russell-square.*

1841 Henry Pitman, M.D., *Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Montague-place, Russell-square.*

1845 George David Pollock, *Curator of St. George's Hospital Museum; Bruton-street, Berkeley-square.*

ELECTED

1843 Charles Pope, M.D., M.A., F.L.S., *Temple Cloud, near Bristol.*
 1846 Jephson Potter, M.D., F.L.S., 13, *Oxford-road, Manchester.*
 1840 Lewis Powell, *John-street, Berkeley-square.*
 1842 James Powell M.B., *Great Coram-street, Brunswick-square.*
 1839 John Propert, *New Cavendish-street.*
 1814 William Prout, M.D., F.R.S., *Sackville-street.* C. 1816.
 V. P. 1823.
 1845 John Pyle, *Oxford-terrace, Hyde-park.*
 1816 Sir William Pym, M.D., *Inspector of Hospitals.*
 1830 Jones Quain, M.D., *Paris.*
 1835 Richard Quain, F.R.S., *LIBRARIAN, Surgeon to University College Hospital, and Professor of Anatomy in University College; Keppel-street.* C. 1838. L. 1846.
 1807 John Ramsey, M.D., *Physician to the Infirmary at Newcastle.*
 1821 Henry Reeder, M.D., *Ridge House, Chipping, Sudbury.*
 1835 G. Regnoli, *Professor of Surgery in the University of Pisa.*
 1842 David Boswell Reid, M.D., *House of Commons.*
 1846 James Reid, M.D., *Physician to the Infirmary of St. Giles's and Bloomsbury; General Lying-in Hospital, &c.; Bloomsbury-square.*
 1847 Samuel Richards, M.D., 39 A, *Bedford-square.*
 1829 John Richardson, M.D., F.R.S., *Surgeon to the Naval Hospital; Chatham.*
 1843 Joseph Ridge, M.D., *Cavendish-square.*
 1845 Benjamin Ridge, M.D., *Putney, Surrey.*
 1817 *John Robb, M.D., *Deputy-Inspector of Hospitals.*
 1821 Charles Julius Roberts, M.D., *Physician to the Adult Deaf and Dumb and Welsh Charity; 31, New Bridge-street.* C. 1827.
 1829 *Archibald Robertson, M.D., F.R.S. L. and Ed., *Physician to the General Infirmary, Northampton.*
 1843 George Robinson, M.D., 40, *Blackett-street, Newcastle-on-Tyne.*
 1845 J. M. Edward Roche, M.D., 12, *Cumberland-street, Portman-square.*
 1843 William Roden, M.D., M.A., F.L.S., *Kidderminster, Worcestershire.*
 1835 George Hamilton Roe, M.D., *Physician to, and Lecturer on Medicine at, the Westminster Hospital; 6, Hanover-square.* C. 1841.
 1836 Arnold Rogers, 296, *Regent-street.*

ELECTED

1846 William Rogers, M.D., *Mortimer-street, Cavendish-square.*
 1819 Henry S. Roots, M.D., *Consulting Physician to St. Thomas's Hospital; 2, Russell-square.* C. 1833. V. P. 1834.
 1829 Sudlow Roots, *Kingston-on-Thames.*
 1836 Richard Roscoe, M.D.
 1835 *Caleb B. Rose, *Swaffham.*
 1845 Henry Mortimer Rowden, *Lecturer on Anatomy at the Middlesex Hospital School of Medicine; 35, Baker-street, Portman-square.*
 1841 Richard Rowland, M.D., *Physician to the Bloomsbury Dispensary; 7, Woburn-place, Russell-square.*
 1837 J. Forbes Royle, M.D., F.R.S., F.L.S., F.Z.S., *Professor of Materia Medica and Therapeutics in King's College, London; 4, Bulstrode-street, Manchester-square.* C. 1846.
 1836 James Russell, *Birmingham.*
 1845 James Russell, Jun., *Birmingham.*
 1827 *Thomas Salter, F.L.S., *Poole.*
 1844 *Thomas Bell Salter, M.D., F.L.S., *Ryde, Isle of Wight.*
 1842 George Sampson, *12, Chester-street, Belgrave-square.*
 1847 William H. O. Sankey, *London Fever Hospital, St. Pancras.*
 1845 Edwin Saunders, *Surgeon-Dentist to, and Lecturer on Diseases of the Teeth at, St. Thomas's Hospital; 13, George-street, Hanover-square.*
 1834 Ludwig V. Sauvan, M.D., *Warsaw.*
 1840 Augustin Sayer, M.D., *28, Upper Seymour-street.*
 1821 Page Nichol Scott, *Norwich.*
 1824 Edward J. Seymour, M.D., F.R.S., *Charles-street, Berkeley-square.* C. 1826. S. 1827. V. P. 1830.
 1840 William Sharp, F.R.S., F.G.S., F.R.A.S., *late Senior Surgeon to the Bradford Infirmary; Humber Bank House, Hull.*
 1837 William Sharpey, M.D., F.R.S. L. and Ed., *Professor of Anatomy and Physiology in University College, London; Gloucester-crescent, Regent's-park.*
 1836 Alexander Shaw, *Surgeon to the Middlesex Hospital; Henrietta-street, Cavendish-square.* C. 1842. S. 1843.
 1839 Thomas Silvester, M.D., *High-street, Clapham.*
 1842 John Simon, F.R.S., *Assistant-Surgeon to King's College Hospital, and Demonstrator of Anatomy in King's College; 11, Wellington-street, Strand.*

ELECTED

1821 Charles Skene, M.D., *Professor of Anatomy and Surgery; Marischal College, Aberdeen.*

1827 George Skene, *Bedford.*

1812 Joseph Skey, M.D., *Inspector-General of Hospitals.*

1824 Frederick C. Skey, F.R.S., *Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Northern Dispensary; Grosvenor-street. C. 1828. L. 1829. V. P. 1841.*

1810 Noel Thomas Smith, M.D., *Newcastle.*

1822 Southwood Smith, M.D., *Physician to the Fever Hospital, and to the Eastern Dispensary; 38, Finsbury-square. C. 1838.*

1835 John Gregory Smith, *Harewood, Yorkshire.*

1838 Henry Smith, *Surgeon to the General Dispensary, Aldersgate-street; 17, Henrietta-street, Cavendish-square.*

1845 William Smith, *Upper Berkeley-place, Bristol.*

1847 William Smith, M.D., *Weymouth, Dorsetshire.*

1843 Robert William Smith, A.M., M.D., M.R.I.A., *Lecturer on Surgery at the Richmond Hospital School of Medicine; Surgeon to the Talbot General Dispensary and Island Bridge Lunatic Asylum; 62, Eccles-street, Dublin.*

1843 John Snow, M.D., *Frith-street, Soho-square.*

1819 *George Snowden, *Ramsgate.*

1816 *John Smith Soden, *Clifton.*

1830 Samuel Solly, F.R.S., *Senior Assistant-Surgeon to St. Thomas's Hospital; Surgeon to the General Dispensary, Aldersgate-street; 1, St. Helen's-place. L. 1838. C. 1845.*

1844 Frederick R. Spackman, M.B., *Harpden, St. Alban's.*

1834 James Spark, *Newcastle.*

1843 *Stephen Spranger, *Swatheling-house, Southampton.*

1838 George James Squibb, 6, *Orchard-street.*

1835 Richard A. Stafford, *Surgeon Extraordinary to His Royal Highness the Duke of Cambridge; Surgeon to the St. Marylebone Infirmary; Old Burlington-street. C. 1840.*

1815 Edward Stanley, F.R.S., *Surgeon to St. Bartholomew's Hospital; 23 A, Brook-street. C. 1821. S. 1824. V. P. 1827. T. 1832. P. 1843.*

1835 Leonard Stewart, M.D., *Keppel-street.*

1842 Alexander Patrick Stewart, M.D., *Physician to the St. Pancras Dispensary; 74, Grosvenor-street.*

ELECTED

1839 Thomas Stone, M.D.

1843 Robert Reeve Storks, *Surgeon to the Blenheim-street Dispensary, and to the Western Dispensary; Gower-street, Bedford-square.*

1844 John Soper Streeter, *Harpur-street, Red Lion-square.*

1827 William Stroud, M.D., 20, *Great Coram-street.* C. 1831.

1847 William Allen Sumner, *Abbey-road, St. John's Wood.*

1839 Alexander John Sutherland, M.D., F.R.S., *Physician to St. Luke's Hospital; Parliament-street.*

1842 James Syme, *Professor of Clinical Surgery in the University of Edinburgh; Charlotte-square, Edinburgh.*

1847 William Symonds, 24, *Blandford-square.*

1844 R. W. Tamplin, *Surgeon to the Orthopædic Institution, Great Queen-street, Lincoln's-inn-fields.*

1840 Thomas Tatum, *Surgeon to, and Lecturer on Surgery at, St. George's Hospital; George-street, Hanover-square.*

1835 J. C. Taunton, *Surgeon to the City of London Truss Society, and to the City Dispensary; 48, Hatton-garden, C. 1840.*

1845 *John Taylor, M.D., *Physician to the Infirmary, Huddersfield.*

1845 Thomas Taylor, *New Bridge-street, Blackfriars.*

1817 Frederick Thackeray, M.D., *Physician to Addenbrooke's Hospital, Cambridge.*

1845 Evan Thomas, *Pwllheli, North Wales.*

1839 Seth Thompson, M.D., *Physician to, and Lecturer on Pathology at, the Middlesex Hospital; 1, Lower Seymour-street.*

1842 Theophilus Thompson, M.D., F.R.S., *Physician to the Northern Dispensary, and to the Hospital for Consumption and Diseases of the Chest; 3, Bedford-square.*

1835 Frederick Hale Thomson, *Surgeon to the Westminster Hospital; Berner's-street.*

1819 John Thomson, M.D., F.L.S., *Physician to the Finsbury Dispensary; 80, Coleman-street. C. 1833. L. 1834.*

1836 John Thurnam, M.D., *The Retreat, York.*

1834 Robert Bentley Todd, M.D., F.R.S., *Physician to King's College Hospital, Professor of Physiology and of General and Morbid Anatomy in King's College; New-street, Spring-gardens. L. 1842.*

1828 James Torrie, M.D., *Aberdeen.*

ELECTED

1843 Joseph Toynbee, F.R.S., *Surgeon to the St. George's and St. James's Dispensary; Argyll-place, Regent-street.*

1808 Benjamin Travers, F.R.S., *President of the Royal College of Surgeons of England; Surgeon Extraordinary to the Queen; Surgeon in Ordinary to His Royal Highness Prince Albert; 12, Bruton-street.* C. 1810. V.P. 1817. P. 1827.

1821 *William Travers, M.D., *Scarborough.*

1841 Matthew Truman, M.D., *44, Gloucester-place, Kentish-town.*

1835 John Cusson Turner, M.D., *Brighton.*

1845 Thomas Turner, *Surgeon to the Royal Manchester Infirmary, and Lecturer on Anatomy; Mosley-street, Manchester.*

1843 William Twining, M.D., *Physician to the North London Ophthalmic Institution; Bedford-place, Russell-square.*

1819 Barnard Van Oven, *Consulting Surgeon to the Charity for Delivering Jewish Lying-in Women; 30, Gower-street, Bedford-square.*

1845 R. A. Varicas.

1806 Bowyer Vaux, *Surgeon to the General Hospital, Birmingham.*

1839 W. R. Vickers, *32, Baker-street.*

1814 John P. Vincent, *16, Lincoln's-inn-fields.* C. 1823. V. P. 1837.

1810 James Vose, M.D.

1846 Alexander Ure, *Surgeon to the Westminster General Dispensary; 24, Bloomsbury-square.*

1828 Benedetto Vulpes, M.D., *Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.*

1841 Robert Wade, *Surgeon to the Westminster General Dispensary; 68, Dean-street.*

1823 William Wagner, M.D., *Berlin.*

1820 Thomas Walker, M.D., *Physician to the Forces, and to the Embassy at St. Petersburgh.*

1821 Tillicard Ward.

1845 T. Ogier Ward, M.D., *Leonard-place, Kensington.*

1846 Nathaniel Ward, *Demonstrator of Anatomy at the London Hospital; 17, Finsbury-place, South.*

1814 Martin Ware, *51, Russell-square.* C. 1844. T. 1846.

1811 John Ware.

1846 James Thomas Ware, *Surgeon to the Finsbury Dispensary; 51, Russell-square.*

1816 *Charles Bruce Warner, *Cirencester.*

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1829 E. T. Warry, *Lyndhurst*.
1837 Thomas Watson, M.D., *Henrietta-street, Cavendish-square*.
1847 *Thomas Watson, *Holbeach, Lincolnshire*.
1818 *Charles Webb, *Oxford*.
1842 Frederick Weber, M.D., *Physician to the St. George's and St. James's Dispensary; Norfolk-street, Park-lane*.
1844 William Wegg, M.D., *Physician to the Westminster General Dispensary; 5, Maddox-street, Hanover-square*.
1842 Charles West, M.D., *Lecturer on Midwifery at the Middlesex Hospital, and Senior Physician to the Royal Infirmary for Children; 96, Wimpole-street, Cavendish-square*.
1841 Thomas West, M.D., *F.L.S., Daventry*.
1840 William Woodham Webb, *Gislingham, near Thwaite, Suffolk*.
1835 John Webster, M.D., *F.R.S., Consulting Physician to the St. George's and St. James's Dispensary; 24, Brook-street. C. 1843*.
1821 Richard Welbank, *Chancery-lane. C. 1826. V. P. 1845*.
1816 Sir Augustus West, *Deputy-Inspector of Hospitals to the Portuguese Forces; Lisbon*.
1828 John Whatley, M.D.
1840 Joseph Wickenden, *Birmingham*.
1824 *William Wickham, *Surgeon to the Winchester Hospital*.
1811 Arthur Ladbroke Wigan.
1844 Frederick Wildbore, *High-street, Shoreditch*.
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1829 Robert Willis, M.D., *Barnes. L. 1838*.
1831 *W. J. Wilson, *Surgeon to the Manchester Infirmary*.
1839 Erasmus Wilson, F.R.S., *Lecturer on Anatomy and Physiology in the Middlesex Hospital, and Consulting Surgeon to the St. Pancras Infirmary; Charlotte-street, Fitzroy-square*.
1839 James Arthur Wilson, M.D., *Physician to St. George's Hospital; Dover-street. C. 1846*.
1825 Thomas A. Wise, *India*.
1841 George Leighton Wood, *Surgeon to the Bath Hospital; Queen-square, Bath*.

ELECTED

1843 John Ward Woodfall, M.D., *Physician to the Western Dispensary; 33, Davies-street, Berkeley-square.*

1847 Robert Woollaston, *Conduit-street, Westbourne-terrace.*

1833 Thomas Wormald, *Assistant-Surgeon to St. Bartholomew's Hospital; Bedford-row. C. 1839.*

1842 W. C. Worthington, *Surgeon to the Infirmary, Lowestoft, Suffolk.*

1835 John Wright, M.D., *Prince's-court, Westminster.*

[It is particularly requested that any change of Title or Residence may be communicated to the Secretaries before the 1st August in each year, in order that the List may be made as correct as possible.]

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Sir David Brewster, K.H., LL.D., F.R.S. L. and Ed., &c., *Cor. Mem. Roy. Institute of France, &c.; Kingussie.*

1841 Robert Brown, D.C.L., F.R.S., *Vice-President of the Linnean Society; British Museum.*

The Very Rev. William Buckland, D.D., F.R.S., *Dean of Westminster.*

1835 William Clift, F.R.S., *Royal College of Surgeons.*

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1841 The Rev. William Whewell, D.D., F.R.S., *Master of Trinity College, Cambridge.*

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1813 Jacob Berzelius, M.D., F.R.S., *Professor of Chemistry in the University of Stockholm.*

Carl Johan Eckström, K.P.S. and W., *Physician to the King of Sweden, First Surgeon to the Seraphim Hospital, Stockholm.*

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Baron A. de Humboldt, *Member of the Institute of France, &c.; Berlin.*

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1841 P. C. A. Louis, M.D., *Physician to the Hôtel Dieu, Member of the Royal Academy of Medicine, &c.; Paris.*

1841 F. Magendie, M.D., *Member of the Institute; Physician to the Hospital of the Salpêtrière; Paris.*

1841 Johann Müller, M.D., *Professor of Anatomy and Physiology; Director of the Royal Anatomical Museum; Berlin.*

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Professor Orfila, *Dean of Faculty, and Physician to the King of the French, &c., &c.; Paris.*

1841 Bartolomeo Panizza, M.D., *Paria.*

1843 Philibert Joseph Roux, *Member of the French Institute; Surgeon in Chief of the Hôtel Dieu; Professor in the Faculty of Medicine; Paris.*

C. J. Temminck, *Director of the Museum of Natural History of the King of Holland; Amsterdam.*

Friederich Tiedemann, M.D., *Professor of Anatomy and Physiology; Heidelberg.*

1841 John C. Warren, M.D., *Professor of Surgery in the Harvard University, Boston, Massachusetts.*

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ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasoning, or opinions, set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its Transactions.

TWO REMARKABLE CASES
OF
ENCEPHALOID DISEASE OF THE HEART,
WITH OBSERVATIONS.

By PRESCOTT HEWETT,

LECTURER ON ANATOMY AT ST. GEORGE'S HOSPITAL MEDICAL SCHOOL.

Received April 20th—Read Nov. 10th, 1846.

As the following two cases present, I think, points of interest not unworthy of consideration, both in a pathological and in a practical view, I trust that they will be acceptable to the Society.

Case 1.—Benjamin Rooth, *aet. 40*, was admitted into St. George's Hospital, under the care of Mr. Keate, on the 15th of May 1844, with a large tumour, apparently of a carcinomatous nature, connected with the posterior part of the left foot. The disease had begun, about a year before the patient's admission into the hospital, by pain in the heel, for which he could assign no cause; this pain was followed by swelling of the part, which, within the last six months, had been rapidly increasing in size.

The patient, although somewhat reduced, stated that he enjoyed good bodily health; his countenance was somewhat pale, his pulse natural, his respiration free, and no œdema was observed about the extremities. At a consultation of the surgeons, it was determined that the leg should be removed, as there were no symptoms of visceral disease. Mr. Keate performed the operation on the 25th of the month, and upon examining the tumour it proved to be of an encephaloid nature, the bones and soft tissues of the back part of the foot

being implicated in the disease, which had apparently begun in the os calcis.

For the first two days after the operation, the patient appeared to be doing very well; but on the third day there was slight irritative fever, and the stump had put on an unhealthy appearance: on the following day he was in a very low state, with an intermittent pulse, and complained of pain in the right hypochondrium. These symptoms went on increasing; jaundice, profuse perspirations and severe pleuritic pain of the left side, made their appearance on the 30th, and the patient died on the 31st.

The body was examined thirteen hours after death. Marks of extensive recent inflammation, of a low character, were found in the left pleura, and in the pericardium. Slight emphysema existed at the anterior part of the lungs, and, at the posterior part, these organs were congested, but they did not present any other morbid appearance. The heart, much increased in size, presented dilatation of the right cavities, with hypertrophy of the muscular structure of the left ventricle. In the right auricle was a large growth, which, proceeding from the appendix, occupied the greater part of the cavity of the auricle, and, passing through the auriculo-ventricular opening, projected into the ventricle, reaching nearly to the bases of the columnæ carnicæ of the valve.

The shape of this growth was conical, with its basis towards the auricle; it distended the cavity of the appendix, in which it appeared to have originated, and it was also connected with that part of the auricle in the immediate neighbourhood of the appendix, but it was impossible to decide which of the tissues of this organ had been the primary seat of the disease. The surface of the tumour was irregular, and its tissue firm, except at the lower part, where it was somewhat flocculent: when cut into, it presented a highly vascular appearance, and in structure resembled exactly the encephaloid disease of the foot, for which the patient's leg had been removed. The valves of the heart were healthy in structure, and all the cavities of this organ contained coagula of blood, for the

greater part fibrinous. No morbid appearance was observed about the viscera of the abdomen. Some enlarged glands were found in the groin, but they did not contain any carcinomatous deposit. The stump presented an unhealthy appearance, and one of the posterior tibial veins, which, at its extremity, was open to the extent of half an inch, contained some pus, above which were coagula, apparently of a healthy nature; the other vessels did not present any morbid appearance. No carcinomatous disease was discovered in any other part of the body.

The growth found in the right auricle was not examined by the microscope, but its general characters were so well marked that I have no doubt as to its encephaloid nature.

Case 2.—Mary Hudson, *aet. 59*, was admitted into St. George's Hospital, under the care of Mr. Keate, on the 10th of December 1845, with a tumour of the right breast, as large as the egg of an ostrich. The disease had existed about six months, and made its appearance after a blow on the breast, connected with a fall. The tumour, which was very painful, presented evident fluctuation in front, but at the back part it was solid; to its outer side was another tumour, of the size of a pullet's egg, firm in structure and apparently connected with one of the absorbent glands; the axillary glands were slightly enlarged; a red blush, of an erysipelatous character, existed in the skin covering the tumour, and had spread to some distance. The countenance was anaemic and of a yellowish colour; the respiration free, and the pulse weak, but otherwise natural. The patient, who was cheerful, presented no symptoms of disease beyond that of the breast.

On the 14th a grooved needle was passed into the front part of the tumour, and a pint of straw-coloured serum was let out; another puncture was then made at the back part of the tumour, but no fluid followed. On the 18th, the breast being as large as before, a fresh puncture was made in the neighbourhood of the first, but this time a small quantity only of blood and serum escaped.

About three weeks after the admission of this patient into the hospital, a consultation of the surgeons was held, at which it was determined, under existing circumstances, not to meddle with the breast. At this time the patient was in good spirits, and there were no symptoms whatsoever to draw attention to any organic disease of any one of the internal viscera. By degrees the tumour gradually increased in size; the skin covering it assumed a duskier hue, and ultimately gave way at the points where the punctures had been made; the ulceration spread, and a large fungating growth made its appearance. She continued in this state, but gradually getting weaker, until the 20th of January 1846, when, at half-past five in the afternoon, she was suddenly attacked with urgent dyspnoea and partial syncope, the pulse at the wrist being scarcely perceptible; these symptoms increased, notwithstanding the various remedies made use of, and she died on the following day at a quarter to one.

At the examination of the body, twenty-two hours after death, the tumour of the breast proved to be of a decidedly encephaloid nature; it was remarkably soft, almost fluctuating, and, in several places, presented the peculiar yellow opaque appearance so commonly observed in these tumours. Several of the absorbent glands were affected; one of them, of the size of a large egg and situated close to the original tumour, presented a well-marked specimen of this disease. A small encephaloid tumour was also found in the left rectus abdominis, the muscular structure of which it had partially destroyed. The cavities of both pleurae contained a quantity of clear serum, which in the left amounted to about a pint; and, on the right side, there were some old adhesions. The lungs were quite healthy, with the exception of trifling emphysema at the anterior part, and slight congestion at the posterior part. The heart was enlarged by dilatation of its cavities, which on the right side were otherwise healthy. The dilatation of the left auricle was more marked than that of the other cavities; the mitral valve was slightly contracted, and there was some thickening of its tendinous chords: the

auricular surface of this valve was, for the greater part, covered over by a soft deposit, pinkish in colour, of an irregular shape, resembling that of a cauliflower, and, to the naked eye, presenting exactly the appearance of the mass of the encephaloid disease observed about the breast and glands. This species of vegetation projected some distance, both into the auricle and into the ventricle, running along the surface of the tendinous chords, and covering the extremities of the muscular columns; and by it the auriculo-ventricular opening was all but clogged up, there being merely an opening, of the size of a quill, left for the passage of the blood. Portions of the diseased structure were easily scraped off with a knife, but the mass of it was firmly connected with the parts upon which it was situated. The aortic valves were slightly thickened, and two of them had, at their apices, become united to each other. No encephaloid disease existed in any other part of the heart, and no morbid appearance was observed about the coagula contained in the cavities of this organ. The abdominal viscera were healthy. No oedema existed about the extremities.

The portions of the diseased structure, both of the breast and of the heart, which were examined by the microscope, presented some large nucleated cells, of a spherical shape, with a quantity of granular matter floating about, but no caudate cells were observed in either of the specimens.

Both the preceding cases present rare forms of carcinomatous affections of the heart, but it is to the latter of them that I am more especially desirous of calling the attention of the Society, as in this case there existed a well-marked specimen of encephaloid disease of the free surface of the endocardium, an affection rarely met with,—so rarely indeed, that Dr. Walshe,* in his elaborate treatise on “Cancer,” lately published, after stating his opinion that “there is nothing repulsive to reason in the idea of limitation of cancerous disease to the endocardium, but that if such cases really occur

* *Nature and Treatment of Cancer*, 1816, p. 371.

they are singularly rare,"—alludes to one case only, which he quotes from the *Revue Médicale*, and even of the true nature of this case he expresses very great doubt; an opinion which is, I think, most fully warranted by the description of the morbid appearances.

The species of vegetation which was found on the mitral valve of Mary Hudson presented, to the naked eye, all the characters of encephaloid disease, and, when compared with portions of the diseased breast and gland, the resemblance was striking. The disease with which such a vegetation was most likely to be confounded is that form of soft fibrinous deposit so commonly observed about the surface of the endocardium, but the microscopical examination of this vegetation at once pointed out that such was not the nature of this deposit. Its general appearance, and the circumstances under which it occurred, led me to think that it was of an encephaloid nature, and the opinion which I then formed was subsequently strengthened by the following observation from Rokitansky:/* "In rare cases, and under the requisite constitutional conditions, the vegetations on the valves of the heart may be of a cancerous, or more especially of a medullary, nature."

I avoid entering into a discussion concerning the origin of the encephaloid disease of the heart, in the latter case, as this would be foreign to my present purpose.

With regard to the first case, it is impossible to decide whether the disease began in the lining membrane of the appendix, or in its subjacent structures; but, whatever its origin, the fact of a tumour of this size, projecting from the right auricle into the ventricle and occupying so large a portion of the auriculo-ventricular opening, without ever producing symptoms sufficiently marked to call attention to the circulating organs, is certainly interesting, and only to be explained by the tumour growing slowly, and thus allowing the parts to accommodate themselves to its presence.

In a practical view, the two cases present, I think, points of

* Vol. ii. p. 507.

great interest. In the first case, the patient underwent a most severe operation, without there being the slightest suspicion of the disease of the heart, no symptom being present which could in any way lead one to suppose that this organ was so seriously affected: every thing went on as usual at such operations, and the only wonder is, that, under so severe a trial, the function of the central organ of circulation was not interfered with, and that the man did not die on the operating table. A case somewhat similar, in which the disease was, however, more extensive, is given by Cruveilhier,* but in this case the man died suddenly after a hearty meal. In the second case, the question naturally arises as to how long the carcinomatous affection of the heart had existed, and whether it existed at the time of the consultation of the surgeons. Such a deposit as that which was found on the mitral valve, in this case, might take place very rapidly, but as the patient did not present, until within a few hours of her death, any symptoms of disease of the heart, it is impossible to say when this disease began.

The preparations illustrative of the two cases which form the subject of this paper, are in the Pathological Museum of St. George's Hospital.

* *Anatomie Pathologique*, liv. xxix.

CASE
OF
TUMOUR IN THE GROIN,
WHERE THE TESTICLE HAD NOT DESCENDED ;
AND
OPERATION FOR ITS REMOVAL.

BY J. MONCRIEFF ARNOTT, F.R.S.,
PRESIDENT OF THE SOCIETY,
SURGEON TO THE MIDDLESEX HOSPITAL.

Received November 24th—Read November 24th, 1846.

RICHARD LONG, 43 years of age, farm servant, admitted into the Middlesex Hospital November 3rd, 1846. In the right groin is a prominent swelling, the size of a man's fist, but of oval shape, the long diameter being nearly in the direction of Ponpart's ligament, which, however, it covers somewhat obliquely ; for whilst its main bulk at the upper and outer end is above the line of the ligament, the larger part of the lower and inner is below it. It extends from within 2½ inches of the anterior superior spinous process of the ilium to a little beyond the pubes, where it comes in contact with the root of the penis, which is slightly displaced to the left side. The skin covering the tumour is of its natural colour and temperature — handling the part gives no pain. The swelling, which has an uniform smooth surface, feels tense and firm, giving generally the idea of solidity, but at one part with an indistinct sense of fluidity. It is not diaphanous. Squeezed in every part, the sensation experienced on compression of the testicle is nowhere felt. The tumour is to a certain extent moveable, but it cannot be raised from its attachments behind, nor on

its abdominal edge ; that is, the finger cannot here get under it, which it can partially elsewhere. No impulse is communicated to the swelling on coughing whilst the hand is placed on the abdominal muscles, and when this is withdrawn the impulse is one of displacement, not of distention ; from the motions observed on its surface during the action of coughing, the tumour must be covered by at least the superficial fascia of the abdomen ; the testicle and scrotum on this side are wanting.

The patient, a fresh-coloured, healthy-looking man, with no other evidence of disease, is the father of seven, and has had thirteen, children,—the last two years ago. He states that he never had a testicle on that side, but that there always was a small swelling, the size of a nut, at the lower part of his belly ; and he points to a situation corresponding to the internal abdominal ring, or upper part of the inguinal canal. Four years ago this swelling came lower down, and was the size of a walnut ; and when he noticed this, although it gave no pain, he tried to get it up, but without success : since then it has continued to enlarge gradually, but without inconvenience, till of late, when its size has interfered with the complete flexion of the thigh in his occupation of making trusses of hay.

Viewing the case as one of disease of the testicle, which had not descended, but being unable to determine its precise nature, whether hydrocele or haematocele with a thickened tunica vaginalis, cystic sarcoma, or malignant disease, I told the patient that it would be necessary to puncture the tumour, and then to proceed according to its nature, so as even to remove it if necessary. Not being prepared for this, and no local applications having been tried, he expressed a wish that they should be, which was done, and at the end of a week he assented to the proposal which had been originally made.

Nov. 13.—The tumour was punctured with a lancet at its lower part, but only blood escaped ; the integuments were therefore divided over its whole length, by which the superficial fascia was exposed, and under this a dense tendinous expan-

sion, that of the external oblique muscle; this being opened, was slit up on the director to the extent of the wound in the integuments, when a purple-coloured glistening tumour burst forth, like the contents of a hernial sac; the tunica vaginalis testis, which was adherent to the neighbouring parts, had been opened by the last incision, exposing the diseased testicle, possessed of its natural relations to the tunica vaginalis, and attached to this posteriorly from the pubes below to the internal abdominal ring above, which was seen quite at the upper and outer angle of the wound. Having divided these attachments, the finger was passed with some difficulty, owing to the close contact of the diseased mass with the internal ring, under the spermatic cord, which was cut across, the vessels being tied as they were divided. The large cavity left by the removal of the swelling had a smooth surface throughout, excepting where the detachment had been effected; in fact, it was lined by the tunica vaginalis, which, as has been already stated, had become united to all the adjoining parts.

It is needless to detain the Society with a description of the dressing of the wound, or with daily reports. It is sufficient to state that up to this time, Nov. 23rd, the case proceeds favourably.

A section of the mass presented the ordinary appearance of medullary sarcoma. No trace of the natural structure of the testicle could be observed; but the vas deferens has been traced by Mr. Moore, on its back part, and with the cord at the line of section presented a healthy character. In the latter, though somewhat enlarged, no encephaloid matter was seen.

A cast of the parts previous to the operation is placed on the table, with the tumour itself, which measures upwards of 5 inches in length, by 3½ from before backwards.

Mr. Curling, in his work on the testicle, remarks that there are "a few instances on record in which a testis retained in the inguinal canal has become so diseased as to lead to the necessity of castration;" and he alludes to a case briefly no-

ticed by Mr. Pott, of diseased testicle in the groin removed by operation at St. George's Hospital. Mr. Curling also refers to two cases by Italian surgeons, and a fourth by a German.

In the Museum of St. Bartholomew's Hospital, there are two casts, which I have recently seen, of tumour in the right groin, where the testicle had not descended. In one of them it was presumed, in the other it was proved to have been encephaloid disease of that organ. In the latter instance, the man, aged 43, had a small tumour in his groin at birth; seven years before his death it began to enlarge; ultimately suppuration took place, with copious haemorrhage from time to time. On examination, *post mortem*, the mesenteric glands were found enlarged and indurated, and, being cut into, discharged the brain-like substance observable in medullary sarcoma. The testicle could not be found; nor could the spermatic cord be traced beyond the tumour, though it was carefully sought for. What remained of the tumour was a mass of soft encephaloid substance.

The following case, probably of undescended and diseased testicle, occurred to my friend Mr. Hodgson, of Birmingham, who has very kindly communicated these particulars of it to me.

"The patient, Mr. M., a respectable farmer, about fifty years of age, had enjoyed good health throughout his life, and was the father of a family. His right testicle had never descended; but remained, as he expressed it, at the rim of his belly. When he first consulted me in 1842, I found a tumour, about as large as a very large hen's egg, in the right inguinal canal. It had formed gradually, and was not at all painful; but when pressed he said that it gave him a sickening feeling, exactly like that which was produced when the other testicle was squeezed. The swelling was firm, but elastic, and smooth to the touch, and moved only with the other parts in which it was imbedded. The left testicle only was in the scrotum; it was healthy. The tumour in the right groin gradually enlarged, and in a few months was

larger than a fist, retaining the same feeling as when I first examined it, and projecting towards the linea alba and forwards. He went through a very long course of iodine, and iodine was used locally, but without the least benefit. Then he went through a full course of mercury and was salivated; but the swelling continued to grow. At length it was evident that a fluid existed in the upper part of the swelling—the lower half being composed of a solid substance. The fluid increased in quantity, and when the tumour was as large as a foetal head it was tapped, and about a pint of straw-coloured albuminous fluid, exactly like that proceeding from a hydrocele, was drawn away. Some of this fluid was sent to Mr. Erasmus Wilson for examination by the microscope, but no spermatozoa could be discovered in it. When the fluid was taken away, the large tumour could be felt smooth on its surface, but extending irregularly upwards into the cyst, from which the fluid had been removed. The accumulated fluid was removed several times by tapping, and on each occasion it had the same appearance, but varied in quantity. The swelling gradually enlarged until the whole tumour became larger than the patient's head. It projected forwards, and extended to the left side, beyond the linea alba, retaining the same characters as formerly, and the same kind of mobility in connection with the adjacent parts. Percussion distinctly showed the boundaries of its fluid and solid components. It was so large and inconvenient that it was necessary to support it by a kind of sling bandage. The general health continued good, and he had no tumour in any other part of his body.

"The swelling was tapped in October 1845, for the last time. The operation was followed by great inflammation of the sac and tumour—gangrene came on, and he died. I am extremely sorry to add that permission, which was anxiously sought, could not be obtained to examine the body.

"My own opinion of the case was, that this tumour was a diseased and undescended testicle. Whether the fluid was contained in a proper tunica vaginalis, or whether a serous

cyst had become developed upon the swelling, may be regarded as doubtful. If the former was the case, the diseased testicle was combined with hydrocele. I thought the disease to be either simple saccocle, or fungoid or hydatid disease of the testicle ; but when its growth continued uninterrupted under the use of full courses of mercury and iodine, I believed that it was not likely to be simple saccocle. The patient was seen by Sir Benjamin Brodie, Mr. Aston Key (who tapped the cyst to enable him and Sir B. Brodie to examine the solid part of the tumour), by Mr. Stanley, Mr. Soden of Bath, and several other surgeons, all of whom I believe coincided with me in the opinion that the case was most probably an undescended and diseased testicle."

The account of Long's case was only brought up to the eleventh day after the operation, on the twelfth he was attacked with erysipelas of the face and head, of which he died in three days. On examination a small deposit of encephaloid substance was found on the right spermatic cord just within the inner ring, and a large mass in the root of the mesentery, which, owing to his being fat, had not been detected during life. There were no deposits in the liver or lungs.

Subsequent to the reading of the above communication to the Society, an account of a similar case has been published in the Medical Gazette, for January 15, 1847, by my friend Mr. Storks. In this instance the tumour was in the left inguinal region, the size of a cocoa-nut, the testicle never having descended. The tunica vaginalis contained fluid, and the testicle was the seat of medullary sarcoma. The removal of the parts was performed by Mr. Storks in January 1846. The patient recovered, and lived till April 1st, 1847, when he died in St. George's Hospital, of the same disease, developed to a great extent in the abdomen.

May 1847.

PENETRATING WOUND OF THE NECK,

ACCOMPANIED BY

PROFUSE ARTERIAL HÆMORRHAGE ;

TREATED SUCCESSFULLY BY LIGATION OF THE COMMON
CAROTID ARTERY.

BY F. LE GROS CLARK, (SEC.)

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Received January 12th—Read January 26th, 1847.

BENJAMIN Wood, a policeman, aged 29, tall, thin, of intemperate habits and bad constitution, was brought to St. Thomas's Hospital, on October 14th, at a quarter before two o'clock. A quarter of an hour previously he had quarrelled with his wife, whilst they were sitting together at dinner, and she hurled a knife at him, which entered his neck on the left side, just below the angle of the jaw and in front of the sternomastoid muscle, making a clean wound about an inch in length. The knife with which the injury was inflicted was such as is commonly used at table, but worn away at the extremity of the blade, so as to present a rather sharp point and a cutting edge on both sides. He afterwards stated that he stanched the blood as well as he was able, whilst he ran down stairs to a surgeon close at hand, who applied a compress to the wound and immediately brought him over to the hospital.

When he was admitted, his clothes were saturated with blood, and he was still bleeding profusely. Mr. Solly, who was on the spot, directed that the common carotid should be compressed, and then enlarged the wound in different directions, in the hope of finding and securing the bleeding vessel or vessels,—as it seemed doubtful whether

the trunk of the external carotid alone was wounded, or whether its branches, and possibly the internal carotid, were not involved in the injury, for arterial blood flowed in profusion in spite of the pressure made on the common trunk. One ligature was applied at a point whence the blood seemed to issue, but without any effect. When I came to the surgery Mr. Solly had just succeeded in controlling the bleeding by pressing his fingers deeply into the wound, in the direction in which the knife seemed to have passed, namely, downwards and inwards.

The patient at this time was blanched and almost pulseless, and had vomited. After conferring with my colleague for a few moments, it was agreed that, as another gush of blood might prove fatal, I should place a ligature on the common carotid, whilst he retained his command of the bleeding vessels. An incision about two inches in length was made along the anterior border of the sterno-mastoid muscle, extending from the thyroid cartilage downwards; and when the superficial structures were divided, care having been taken to avoid injuring the external jugular vein and some branches of the cervical plexus, the sterno-mastoid and omo-hyoid muscles were held aside by an assistant with a retractor. The descendens lingualis was gently drawn to one side, and a small opening was made in the sheath of the vessels, which brought the artery into view. An aneurism needle was passed round the pulseless artery, and neither the par vagum nor internal jugular vein was seen. A large twisted silk ligature was used; but was not instantly tightened on account of a fit of vomiting which came on at the moment of passing the needle. Scarcely a tea-spoonful of blood was lost during the operation; and after the knot was made and the pressure entirely removed, not a drop escaped from the original wound. A single suture and two strips of adhesive plaster were employed to close the lower wound, and the upper one was plugged with a piece of sponge wrapped in gauze. The patient was then removed to bed.

Vespere.—Appears comfortable, and has slept half an

hour. Pulse feeble and rapid. Liq. Opii. sedat. mxx. si opus sit.

15th.—The anodyne was given, which procured a quiet night with refreshing sleep. I removed the compress and ligature which had been applied at the upper wound; and, as no bleeding followed, I brought its edges towards each other with strips of plaster. Pulse about 96, and improved in power. Complains of difficulty in deglutition. Diet to be limited to arrow-root and milk.

16th.—Had a quiet night, without opiate. No head symptom nor loss of temperature or sensibility on the left side of the head: facial, temporal arteries, &c., on the left side, entirely pulseless. The difficulty of deglutition has increased, accompanied by accumulation of mucus in the air-tubes. The suture was removed from the lower wound; and he was permitted to have beef-tea in small quantities.

17th.—The anodyne was repeated last night. Uvula and palate much inflamed, and dysphagia very distressing: says he feels comfortable in other respects. Upper wound sloughy. Bowels relieved by enema. Pulse 92, even and of moderate power.

19th.—Improving; countenance better; he has some appetite, and can swallow with less effort and pain; but is obliged to relieve the sensation of suffocation by forcible expulsion of the mucus from the windpipe. Upper wound granulating, and producing a copious sanguous discharge; lower wound has re-opened, but looks healthy. Pulse 96, quiet. Bowels relieved by injection. Takes the opiate at night.

21st.—He feels stronger; but his cough is troublesome. Wounds healthy; heart's action feeble, but pulsation in sub-clavian and carotid arteries strong.

24th.—Free discharge from both wounds; and there appears to be a sinuous communication between them: a compress to be applied in the intervening space. Takes one egg besides his beef-tea daily.

28th.—Not so well: has a hot skin, thirst, and rapid pulse; wounds a little foul, and granulations large and florid. The

latter were touched with sulphate of copper, and the wounds were lightly strapped and covered with water-dressing. Castor-oil and fever mixture ordered. The ligature being still firm, it was put on the stretch, and fixed down by a piece of adhesive plaster, so as to keep up a constant and gentle traction on it.

On the day following the above report, the ligature came away without the employment of any force, and the health of the patient subsequently improved. The purulent discharge continued rather abundant for some time; but the compress prevented its burrowing from one wound to the other. There was also a considerable quantity of limpid secretion from the upper wound, probably supplied by the parotid gland which must have been involved in the injury. His diet was gradually improved.

On the 4th of December he caught cold, and had a fresh collection of matter at the lower part of the neck; this subsequently escaped by the lower wound, which was not entirely healed. He is now (December 31st) quite well. When he left the hospital there was no return of pulsation in the branches of the external carotid.

The interest in the foregoing case is principally of a negative kind, consisting in the entire absence of any untoward symptom associated with, or arising from, the operation. It would seem, therefore, to confirm the opinion that, in similar penetrating wounds in this situation, followed by arterial haemorrhage threatening life, it is better at once to place a ligature on the common carotid than to lose time in searching for the bleeding vessels. A satisfactory sequel in the present instance was less anticipated, as the patient had a constitution undermined by previous ill-health and irregular habits; moreover, before the separation of the ligature he suffered from severe catarrhal sore-throat, accompanied by bronchitis,—a serious complication, as it involved the necessity of frequently making a strong effort to expel the mucus which clogged the air-passages.

This case further presents an illustration of a remark

which occurs in Dr. Burrows' recent work on "Disorders of the Cerebral Circulation," that cerebral symptoms rarely supervene where there has been considerable loss of blood prior to the application of a ligature on the trunk of the common carotid artery. In the present instance the external carotid could not have been secured without an extension of the original wound; and even in the event of the internal carotid proving to be intact, the ligature must most probably have been applied close to the point of bifurcation of the common trunk.

A day or two after the operation I observed that the left angle of the mouth was perceptibly raised above the right, and that the lips came first in contact on the same side in closing the mouth. This peculiarity continued throughout the case, and it was not until a considerable time had elapsed that I was able to satisfy myself that the sound side was not partially paralysed: but, as the patient distinctly possessed more power in retracting the right than the left commissure of the mouth, I suspect that the phenomenon alluded to was partly attributable to natural expression, and partly the consequence of inflammation extending to the trunk of the left facial nerve, from its contiguity to the original wound.

SOME INSTANCES OF THE CONTRAST BETWEEN
DELIRIUM TREMENS
AND
INFLAMMATION OF THE BRAIN,
AS REGARDS THE
QUANTITY OF PHOSPHORIC ACID EXCRETED BY THE
KIDNEYS.

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Received December 17th, 1846—Read January 26th, 1847.

PHOSPHORIC acid is excreted combined with the earths and with the alkalies. If lime or magnesia be present in sufficient quantity, then the whole of the phosphoric acid present in the urine is precipitated, on the addition of ammonia, in combination with these earths: but if an insufficient quantity of earthy matter be present, then the excess of phosphoric acid remains combined with potash or soda. Usually the lime and magnesia excreted by the kidneys are not equal to combine with above $\frac{1}{10}$ th part perhaps of the phosphoric acid in the urine. From $\frac{1}{4}$ th to $\frac{1}{8}$ th will be earthy phosphate, and the other $\frac{3}{4}$ ths to $\frac{7}{8}$ ths alkaline phosphate. Hence the variations in the quantity of earthy phosphate precipitated by ammonia will usually depend on the quantity of lime or magnesia passing through the system; and the sum of the alkaline and earthy phosphates must be determined if we desire to arrive at a knowledge of the quantity of phosphoric

acid passing out of the system. In an extended inquiry* into the highest amount of phosphates excreted, it might have been anticipated that the determination of the lowest limit would be of equal interest with that of the highest; but very many analyses were made before the diminution of the phosphates in some cases of disease was made evident.

The following cases of delirium tremens present the lowest limit to the amount of phosphates; being in the lowest instance only .06 per 1000 urine, specific gravity 1017.9: whilst the cases of inflammation of the brain present the higher limit to the amount of phosphates excreted; being in the highest 13.38 per 1000 urine, specific gravity 1031.1: that is, 223 times less in the former case than in the latter, so far as a comparison may be made, the specific gravity and quantity of the urine not being taken into the account; and this comparison is the more admissible inasmuch as though the specific gravity of the urine is for the most part highest in inflammation of the brain, yet the quantity of urine in delirium tremens is usually excessively diminished.

It will be seen that in about twenty hours, in one case of inflammation, 58 ounces of water, specific gravity 1024.8, were excreted; whilst in a case of delirium tremens, in eleven hours the whole quantity excreted was 4 ounces, specific gravity 1019.1.

In the three cases which are here related of inflammation, the average quantity of phosphates is 8.26 per 1000 urine, specific gravity 1025.3.

During the acute stage of the three cases of delirium tremens, the average quantity of phosphates is only .67 per 1000 urine, specific gravity 1020.4.

Hence, then, sometimes at least, an analysis might determine whether delirium tremens or inflammation of the brain be present.

But it is not every case of delirium tremens which exhi-

* For the mode of analysis, see *Philosophical Transactions*, 1815 and 1846.

bits this diminution; and it is not every case of inflammation of the brain which shows an increase of the phosphates. For if food can be taken in delirium tremens, the food furnishes phosphates, which hinders the diminution from being apparent; and if the inflammation of the brain be slight, the limits of variation in the healthy state rise so high that the slight increase, if present, cannot necessarily be considered as the result of inflammation. So that the chemical analysis can only be regarded as an assistance to the diagnosis, and not as alone sufficient to determine what the disease, in doubtful cases, may be. Such, at least, is the result so far as my experiments have hitherto gone.

The peculiar nature of the urine in delirium tremens is not less interesting to the physiologist than it is to the pathologist. Very frequently thick, scanty and high-coloured, it presents the characteristics of the most intense inflammation; and when it is found that such urine may be acid for a week though containing almost no phosphoric acid, and that it may contain so little phosphoric acid that this in one case was found to be insufficient to combine with above $\frac{1}{27}$ th of the earthy matter which was passing out of the system, it is probable that a more minute attention to such a state of secretion may lead to a more perfect understanding of the nature of the disease.

My intention, however, in the present paper is only to exemplify, 1st, that diminution, in the total amount of phosphatic salts excreted, which is to be observed in some cases of delirium tremens; and, 2ndly, to contrast therewith that increase which, in some cases of inflammation of the brain, is found to exist.

If any quick way of determining the amount of phosphates were possible, it would, without doubt, save much trouble, and add much to the usefulness of the distinction which I consider so diagnostic; but where quantities are in question we can judge only by weight or measure. Now, the measuring of the amount of a precipitate is always inaccurate; and in the case of the amount of phosphates precipi-

pitated is not to be trusted even for rough approximations. Even the balance, from the difficulty of obtaining the substance to be weighed perfectly pure, gives only fine approximations. Hence all determinations of the quantity of phosphates present in the urine must be made with the balance; and the greater the care to obtain the substance to be weighed pure, the more valuable will be the results.

Case 1.—Ann Jones, *aet. 32*, Queen's Ward, under Dr. Nairne, admitted on the 20th of May 1845, with delirium tremens, which had existed about four days, having had no sleep for two nights. She had a draught with a quarter of a grain of morphia every two hours. The following day she had had no sleep: she was not violent, but was constantly getting up in bed. She took forty drops of laudanum instead of the morphia. She continued the opium until she had taken five doses; half an hour after the last dose she went into a fit. She had been very violent previously; she bit her tongue, was convulsed, and then fell asleep for three-quarters of an hour. She awoke very violent; but after some strong brandy-and-water she fell asleep and had a very quiet night, with much sleep.

The water passed in the afternoon was very small in quantity, acid; very thick on standing, from a brick-red sediment; filtered, specific gravity = 1028.7.

33.310 Grammes precipitated by Chloride of Calcium and Ammonia
gave .036 Phosphate of Lime.

Hence Phosphate of Lime = 1.07 per 1000 urine.

22nd.—Manner now more quiet; much less tremor; face injected; pulse 90, small; pupils small, but not excessively contracted. The opium was omitted, and she went out well on the 10th of June.

This case contains only a single analysis, but it shows a remarkable diminution of the phosphates; whilst the specific gravity of the urine is as high as it usually is in inflam-

mation of the brain. The quantity of water being in this case also very much diminished, a very small amount of phosphates was excreted during the time that the symptoms were most severe.

Case 2.—David Davis, an habitual drunkard, ætat. 35, hair-dresser, Hope Ward, under Dr. Nairne, admitted on the evening of the 18th of June 1845, having been in a sleepless state for three days. Reported to have had some anasarca for six weeks.

Two hours after admission he had a fit which lasted ten minutes; snorted and was convulsed; did not fall asleep for nearly an hour, during which time nothing could be made of him. At ten at night he had half a grain of morphia.

19th.—Very quiet during the night. Quiet this morning. Face injected. Answers sensibly; much tremor; urine was made at six in the morning, none having been passed before since seven in the evening; the whole quantity was four ounces, which, on cooling, gave a very pink sediment; when filtered, specific gravity = 1019·1.

65·228 Grammes precipitated by Ammonia } = ·013 = 0·19 per 1000.
—Earthy Phosphate }

32·999 Grammes precipitated by Chloride of } = ·081 = 2·45 per 1000.
Calcium & Ammonia—Phosphate of Lime }

Towards evening he began to be very restless; up and out of bed, and running about. Had no sleep and no food.

20th.—Extreme excitement all night. To-day very restless and excited. Urine passed from six in the evening till six the next morning, about seven ounces; specific gravity = 1019·3. A trace of albumen and few blood globules. Fibrinous cylinders uncertain.

49·949 Grammes precipitated by Ammonia } = ·002 = ·04 per 1000.
—Earthy Phosphate }

33·007 Grammes precipitated by Chloride of } = ·005 = ·15 per 1000.
Calcium & Ammonia—Phosphate of Lime }

33·002 Grammes precipitated by Chloride of } = ·004 = ·12 per 1000.
Calcium & Ammonia—Phosphate of Lime }

21st.—Confined all day; very violent; water drawn off at twelve; whole quantity, from six in the evening, about six ounces; pink sediment not so much as hitherto, strongly acid, and remained so for a week in an open vessel. Specific gravity = 1017.9.

32.961 Grammes precipitated by Chloride of }
Calcium & Ammonia—Phosphate of Lime } = .002 = .06 per 1000.

20.170 Grammes precipitated by Chloride of }
Barium and Nitric Acid—Sulph. of Baryta } = .116 = 5.74 per 1000.

22nd.—In the evening he had two fits, and died.

Examination 15½ hours after death.—There were old and very extensive adhesions uniting the right lung to the sides of the chest. The lung itself, throughout the whole of its extent, was loaded with large quantities of red frothy serum, except at the back part of the lower lobe, where there was red hepatization and softening of texture. The left lung also was loaded, towards the back part, with red frothy serum, which ran out in large quantities when the lung was cut into. In both upper lobes there were, on the surfaces of the lungs, depressions with puckered margins, where the tissue of the lung was condensed and contained several tubercular deposits close to the depressions; but no tubercular matter was found elsewhere. The heart was pale in colour and soft in texture; all its cavities were dilated, but without hypertrophy of the walls. Both the aortic and mitral valves were slightly thickened and somewhat contracted; the other valves were healthy. The blood contained in the cavities of the heart was fluid and very thin. A few patches of atheromatous deposit existed at the root of the aorta.

The cavity of the peritoneum contained a small quantity of dark coloured serum. The liver was of a fawn colour throughout its whole extent. Its margins were round, and its surface presented a well-marked, granular appearance. When cut into, the structure of this organ presented a variegated colour from the condensation of the cellular tissue, which in several places had separated the acini and partially

compressed them. But the condensation of the cellular tissue was not very great.

Both kidneys were large, much congested on their surfaces, and lobular; their capsules peeled off easily, and their surfaces presented, in several places, slight depressions, as if the cortical structure had there partially disappeared. In the other parts, the surfaces of these organs were perfectly smooth. The other organs in the abdominal cavity presented nothing remarkable.

The dura mater was more than usually adherent to the calvaria. The sub-arachnoid cellular tissue and the pia mater were filled with large quantities of clear fluid. The cineritious substance was pale, and the white substance presented slight venous congestion. The ventricles were not enlarged. The structure of the brain appeared to be pretty healthy.

For this and all the *post-mortem* accounts I am indebted to Mr. Prescott Hewett.

This case is a more severe one than the preceding; the patient dying probably about the sixth day of the attack. The phosphates were found gradually to become less and less in quantity, until at last only .06 per 1000 urine, specific gravity 1017.9, were excreted. This is the smallest amount I observed in any of my experiments. The acidity of the urine, when scarcely any phosphoric acid was present, is worthy of remark. Here also is a good example of the diminution of the specific gravity, and of the quantity, of water made.

In eleven hours, 4oz. of water,	specific gravity 1019.1
In twelve hours, 7oz.	„ „ „ 1019.3.
In eighteen hours, 6oz.	„ „ „ 1017.9.

How far this is a conjoint effect of the delirium tremens, of disease of the liver, and of opium, I am not yet able to determine.

The following case shows a diminution of the specific gravity and of the amount of phosphates closely corresponding; and yet in it only 25 drops of laudanum had been taken when the analysis was made.

This second case of delirium tremens is also very interesting, inasmuch as it closely agrees, as to the state of the lungs and the age of the patient, with the second case of inflammation of the brain which I am about to relate; yet how different is the amount of phosphates excreted. Two hundred and nineteen times as much in the latter case, if we compare the greatest quantity of the phosphates excreted in the one case with the least quantity in the other.

Case 3.—George Smart, aet. 35, Fuller Ward, under Dr. Seymour. Admitted early in the morning of July 24th, 1845, having, at three, tried to strangle himself. At seven he slept about an hour, with 25 drops of laudanum. He has had six attacks of delirium tremens previous to this. The present began on the 19th: the patient having been drinking gin for three weeks, and having taken scarcely any food. Senna and salts were given on his admission. Water passed at one o'clock of deep reddish colour, about six ounces, acid. Traces of blood globules, albumen and fibrinous cylinders. Specific gravity = 1018·0.

65·292 Grammes precipitated by Ammonia } = ·007 = ·10 per 1000.
 —Earthy Phosphate }
 32·965 Grammes precipitated by Chloride of } = ·003 = ·09 per 1000.
 Calcium & Ammonia—Phosphate of Lime }
 32·965 Grammes precipitated by Chloride of } = ·407 = 13·34 per 1000.
 Barium and Nitric Acid—Sulph.of Baryta }

To the clear ammoniacal fluid, from which all earthy phosphate had been precipitated, acid phosphate of soda was added. A very plentiful precipitate fell, which, under the microscope, was recognised to be chiefly phosphate of ammonia and magnesia. Thus proving the presence of magnesia:—

The 65·292 Grammes of the Ammonial fluid precipitated by Acid Phosphate of Soda, gave Earthy Phosphate = ·190.

25th.—Has dozed a little. No sleep. Medicine was repeated every four hours. Has taken mutton and potatoes. Pulse 100, soft. Tongue furred, moist. Bowels acted twice last evening. Much perspiration. Very restless, continu-

ally out of bed. Delirious. Urine said to have been plentiful during the night. This day the urine is acid, deep coloured. Specific gravity = 1022.5.

65.564 Grammes precipitated by Ammonia } = .052 = .79 per 1000.
—Earthy Phosphate }

33.111 Grammes precipitated by Chloride of } = .202 = 6.10 per 1000.
Calcium & Ammonia—Phosphate of Lime }

26th.—Pulse very weak. Tongue furred. Pupils very small. Perspiration excessive. But little sleep. Forty drops of laudanum every six hours.

27th.—Slept last evening and all night. Water drawn off at twelve; deep colour, acid. Gave a very considerable precipitate of urate of ammonia; filtered. Specific gravity = 1027.9.

33.284 Grammes precipitated by Chloride of Calcium and Ammonia—
Phosphate of Lime = .280 = 8.11 per 1000 urine.

The following day, specific gravity = 1022.9.

57.554 Grammes precipitated by Ammonia } = .052 = .90 per 1000.
—Earthy Phosphate }

33.122 Grammes precipitated by Chloride of } = .231 = 6.97 per 1000.
Calcium & Ammonia—Phosphate of Lime }

He remained in the house till the 20th of August, quite free from all delirium, tremor, or restlessness, suffering only from an injury.

In this case, examination of the phosphates was made when only 25 drops of laudanum had been taken, yet the amount was diminished to .10 per 1000 urine, specific gravity 1018.0. So little phosphoric acid and so much magnesia were present, that $\frac{1}{7}$ th part only of the magnesia in the urine was precipitated on the addition of the ammonia. From the quantity of sulphuric acid present, and from the patient having taken salts and senna, the greater portion of this excess of magnesia doubtless came from the medicine.

The following day food was taken, and then the amount of phosphates increased, and the urine began to lose the characteristics of delirium tremens.

This case, taken with the previous one, shows the course which the variations in the phosphates follows. As the disease increases, no food being taken, the phosphates diminish more and more. As the case improves, and food is taken, the phosphates rise until the healthy amount is excreted.

In the second case of inflammation of the brain scarcely any food was taken, certainly not more than in the second case of delirium tremens ; and yet the phosphates will be seen passing off in larger quantities than in the healthy state, when food and exercise can be taken.

Case 4.—James Mileham, ætat. 28, waiter, admitted under Dr. Nairne, Jan. 29, 1845, into King's Ward, having been ill about a fortnight, and having had pectoral symptoms for a year.

30th.—Tongue was thickly furred, dry and brownish. Face very red. Skin hot and dry. Pulse 70, full, soft. Pupils rather large, the right a little the larger. Bowels confined. Answers slowly, sometimes rightly, sometimes wrongly ; says he sees but one thing at a time now, but did see double. Seldom answers questions the first time of asking. Head very hot. Delirium during the night. Water was passed at 7 a. m. before any food was taken ; deep-coloured, clear, acid ; gave a precipitate with heat, which re-dissolved with acid. Specific gravity 1025.3.

66.389 Grammes precipitated by Ammonia } = .060 = .90 per 1000.
—Earthy Phosphate }

33.202 Grammes precipitated by Chloride of } = .225 = 6.83 per 1000.
Calcium & Ammonia—Phosphate of Lime }

33.202 Grammes precipitated by Chloride of } = .227 = 6.83 per 1000.
Calcium & Ammonia—Phosphate of Lime }

31st.—Skin and head hot. Pulse 90, full and strong. Tongue very coated, brown, dryish. Face red. Pupils as yesterday. Takes very little food. Looks less heavy. Urine passed at 11 a. m., clear, strongly acid, gave a precipitate of urate of ammonia on standing. Specific gravity = 1028.1.

66.606 Grammes precipitated by Ammonia } = .099 = 1.48 per 1000.
 —Earthy Phosphate }

33.291 Grammes precipitated by Chloride of } = .330 = 9.91 per 1000.
 Calcium & Ammonia—Phosphate of Lime }

33.290 Grammes precipitated by Chloride of } = .326 = 9.79 per 1000.
 Calcium & Ammonia—Phosphate of Lime }

Feb. 2nd.—Pulse 92, much smaller. Tongue brown, dry. Face still flushed. Pupils more dilated. No strabismus. Feeling about for things, and wandering. No sleep at all. Talking, singing and spitting. Much more heavy, and seemingly incapable of answering. Urine passed at 2½ p. m., strongly acid. Specific gravity = 1024.8. Continued acid for a month.

66.328 Grammes precipitated by Ammonia } = .068 = 1.02 per 1000.
 —Earthy Phosphate }

33.184 Grammes precipitated by Chloride of } = .280 = 8.43 per 1000.
 Calcium & Ammonia—Phosphate of Lime }

3rd.—Face not so flushed. Right eyelid more drooping than left. Right pupil more dilated than left. Some strabismus. Pulse small, rapid, 120. Has been roaring all night. Skin hot and dry. Tongue dry and brown. Water was passed last evening. At 2½ p. m. to-day, fifty-eight ounces of urine were drawn off, clear, strongly acid. Specific gravity = 1021.2. Continued acid for three weeks.

66.086 Grammes precipitated by Ammonia } = .065 = .98 per 1000.
 —Earthy Phosphate }

33.068 Grammes precipitated by Chloride of } = .191 = 5.77 per 1000.
 Calcium & Ammonia—Phosphate of Lime }

4th.—Tongue very coated, protruded to the left side. Pulse 120, small. Skin hotter than yesterday. Face flushed, mouth dry. Strabismus more than yesterday. Right pupil the most dilated. More quiet, less wandering. Urine drawn off at 10½ a. m., about 24 ounces, acid to test paper, clear. Specific gravity = 1016.9.

65.670 Grammes precipitated by Ammonia } = .011 = .62 per 1000.
 —Earthy Phosphate }

32.930 Grammes precipitated by Chloride of } = .140 = 4.25 per 1000.
 Calcium & Ammonia—Phosphate of Lime }

Urine drawn off in the evening, about 14 ounces, gave a plentiful deposit on standing. Specific gravity = 1024.6, acid.

66.312 Grammes precipitated by Ammonia
—Earthy Phosphate } = .073 = 1.10 per 1000.

33.177 Grammes precipitated by Chloride of
Calcium & Ammonia—Phosphate of Lime } = .235 = 7.08 per 1000.

5th.—Quite quiet, no wandering. Knew his brother this morning. Pulse 160, not so small as yesterday. Tongue very brown and dry, but put out when the patient was told. Right pupil dilated to the uttermost. He took some tea at 3 P. M., which he asked for, became a great deal more heavy, and died at 7 P. M.

Examination eighteen hours after death.—Membranes of the brain perfectly healthy, on the upper and lateral surfaces; but at the base, especially about the floor of the third ventricle and the fissure of Sylvius, the sub-arachnoid cellular tissue and the pia mater were very much thickened by an effusion of yellow, concrete, semi-transparent lymph. Convolutions of the brain flattened, and sulci somewhat effaced. Both the grey and the white substances of the brain were of a decidedly pink colour. This discolouration of the grey substance existed principally in that part which is next to the white substance. Whilst the discolouration in the latter substance was disposed in bands and streaks, giving the white substance a marbled appearance. Ventricles of the brain distended by a very large quantity of transparent fluid. Septum broken down and flocculent in about its middle; but the neighbouring parts of the fornix and the corpus callosum were certainly not softer than natural. Arteries at the base of the brain healthy.

There were old and firm adhesions partially uniting the right lung to the walls of the chest. On the left side the adhesions were universal and very firm. Both lungs were, throughout their whole structure, thickly studded with miliary tubercles and semi-transparent grey granules; the intervening structure being much congested and somewhat softer than natu-

ral. In different parts of the lungs were several vomicæ of various sizes, the largest being in the apex of the right lung. Pericardium, heart, and coagula, healthy.

Liver and spleen healthy. Two or three small tubercles were found in the cortical structure of the kidneys, which were otherwise perfectly healthy. Several ulcerations destroying the mucous membrane were found in the caput coli; but the other parts of the intestines presented nothing worth noticing.

This case, dying about the 22nd day, is by no means a marked example of the most acute inflammation of the brain. The amount of the phosphates and the specific gravity of the urine are rather high; but the most striking difference from delirium tremens is in the amount of water secreted. In about eighteen hours there were 58 ounces of water, containing as much phosphates as 5.77 per 1000 urine, specific gravity 1021.2. In the succeeding thirty hours at least 38 ounces of urine were drawn off.

The occurrence of inflammation of the brain, in phthisical patients, quite unconnected with tubercles in the brain, although stated and illustrated by Dr. Abercrombie, has not frequently occurred to M. Louis. In two years I have seen in St. George's Hospital seven instances of such a complication. They were all patients about the age of this man; all were examined after death; and the existence of tubercles in the lungs, and no tubercles in the brain, was proved.

Case 5.—John Hitch, brickmaker, ætat. 36, very intemperate, admitted July 17, 1844, into Hope Ward, under Dr. Nairne. Had been for two months an out-patient for cough and spitting. Has complained of his head, and been light-headed for eight days. The face is flushed, the tongue furred, and the pulse slow. Want of all respiration, with dullness at the back of the right lung, and moist rales, and want of healthy respiration at right apex. He was with difficulty induced to take medicine.

20th.—Pulse 60, large and compressible. Seems heavy,
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but does as he is told. Lies on his back. Takes no food, only porter.

The following day he was quite insensible; not answering and picking the bed-clothes. Some strabismus. Urine drawn off, deep coloured, acid, deposited much urate of ammonia: filtered, specific gravity = 1029.7.

66.684 Grammes precipitated by Ammonia } = .122 = 1.82 per 1000.
—Earthy Phosphate

66.684 Grammes precipitated by Chloride of } = .877 = 13.15 per 1000.
Calcium & Ammonia—Phosphate of Lime }

22nd.—Still strabismus. Left pupil dilated, right contracted. Takes no notice; lies on his back, breathing hard. Pulse at times 65. Passes his evacuations in bed. Muttering delirium.

23rd.—Pulse varying from 70 to 85. Strong beating of the carotids. Ptosis of the left eye-lid. The right eye seems to be moved easily, the left eye remains fixed. Moves both his legs and arms. Excessive perspiration. Water drawn off at 10 a. m., deep coloured, acid, no sediment. Specific gravity = 1033.0.

66.900 Grammes precipitated by Ammonia } = .155 = 2.31 per 1000.
—Earthy Phosphate

33.443 Grammes precipitated by Chloride of } = .405 = 12.11 per 1000.
Calcium & Ammonia—Phosphate of Lime }

25th.—Tongue dry. Pulse 120, large, soft and regular. Face very flushed. Left eye quite closed, and pupil immovable; right eye injected. Lies muttering. Face more wasted. Urine deep coloured, acid. Specific gravity = 1030.0.

66.708 Grammes precipitated by Ammonia } = .090 = 1.35 per 1000.
—Earthy Phosphate

33.351 Grammes precipitated by Chloride of } = .318 = 9.53 per 1000.
Calcium & Ammonia—Phosphate of Lime }

26th.—Face less red. Breathing harder. Right pupil more dilated. Pulse 118, not so strong, regular. Much muttering. Tongue dry, brown. Sordes on the teeth. Back beginning to slough.

27th.—In the evening he died.

Examination thirty-eight hours after death.—On the upper part of the head there were some adhesions of the opposed surfaces of the arachnoid, rendering the separation of the dura mater difficult. The structure of the brain was peculiarly firm and tenacious; especially the upper portions of both hemispheres. The ventricles of the brain were much distended with transparent serum. The cerebral substance forming the walls of both lateral ventricles was much softened for the depth of a couple of lines. The fornix and septum lucidum especially were so soft that their natural structure could no longer be recognized. Towards the lower part of the anterior lobe, upon both sides, the medullary substance of the brain also was broken down, and some portions had assumed the consistence of thick cream. The arachnoid membrane at the base and over the pons was rendered opaque by the effusion of serum into the cells of the pia mater. The pia mater generally was congested.

The apices of both lungs were condensed, and presented several small, oval, chalky deposits, mixed with numerous small masses of tubercular deposit. On the right side were old adhesions of the pleura costalis and pulmonalis, between which recent effusions of serum and lymph had taken place.

This is another more acute case exemplifying the connection between phthisis and inflammation of the brain. The quantity of phosphates and the specific gravity of the urine were remarkably increased: 13.15 per 1000 urine, specific gravity 1029.7; and 12.11 per 1000 urine, specific gravity 1033.0. This forms a most marked contrast with the second case of delirium tremens. The smallest possible quantity of food was taken by this patient, so that the amount of phosphates must be considered as uninfluenced thereby.

Case 6.—John Esterling, $\text{ætat. } 36$, stableman, Fuller Ward, under Dr. Seymour, admitted June 28, 1846. Said to have had a cough nine months; to have been ill six weeks, and bad in his head nearly three; complaining of pain and giddiness.

On admission, very stupid and stubborn; knew no one; complained of pain in his head, especially the back part. Face flushed. Sleeping constantly; could scarcely be awoke to take anything; and when he did so was constantly sick. Sees distinctly. Pulse jerking, 44. Has eaten nothing for two days.

C. C. nuchæ ad 3xii. Cal. gr. iij. Antim. gr. iij. Pil. Saponis C. gr. ij.
6tis horis. After cupping, the pulse fell to 40.

30th.—Mouth was very sore, and since it has become so he has not slept so much, and has recognised his wife. Pulse 54. More easily roused. Still constant sickness. Small quantity of urine passed about 7 p.m.: specific gravity = 1031.1, acid, giving a thick pinkish deposit.

65.889 Grammes precipitated by Ammonia } = .125 = 1.89 per 1000.
—Earthy Phosphate }

33.389 Grammes precipitated by Chloride of } = .447 = 13.38 per 1000.
Calcium & Ammonia—Phosphate of Lime }

July 1st.—Omit Calomel. No sickness until the evening; then only once.

2nd.—Face flushed. Pulse 70, compressible, jerking. Doubtful whether he sees correctly. Little difficulty of hearing. Tongue furred. Mouth very sore. Skin cool. Head hot. Sleeps quite quietly, without wandering; but talks and laughs to himself. Passed his motions in bed during the night. More rational to-day than he has been since his admission. Ten ounces of water passed at 7, slight deposit, acid: filtered, specific gravity = 1022.9.

65.188 Grammes precipitated by Ammonia } = .110 = 1.67 per 1000.
—Earthy Phosphate }

33.123 Grammes precipitated by Chloride of } = .200 = 6.03 per 1000.
Calcium & Ammonia—Phosphate of Lime }

3rd.—Much more rational.

5th.—Emp. Lyttæ inter scapulas.

8th.—Pulse 90. Skin cool. Still a little pain in the head.

9th.—Pulse soft, 62. Has been up. Urine passed early in the morning, acid, clear, yellow colour. Specific gravity = 1016.3.

64.305 Grammes precipitated by Ammonia }
—Earthy Phosphate } = .093 = 1.44 per 1000.

32.909 Grammes precipitated by Chloride of }
Calcium & Ammonia—Phosphate of Lime } = .092 = 2.79 per 1000.

From this time he rapidly improved, and went out in his ordinary state of health on the 16th of August.

In this case there were also pectoral symptoms, but the existence of tubercles could not be established by the stethoscope. Soon after his admission, when the symptoms were very urgent, the amount of phosphates and the specific gravity of the urine were much increased; as the symptoms yielded to treatment the phosphates decreased. Taken in connection with the two preceding cases, this points out the course which the variations of the phosphates observe in inflammation of the brain.

Conclusion.

Such then is the contrast which delirium tremens and inflammation of the brain sometimes exhibit as regards the amount of phosphates excreted.

A most marked diminution occurs in the one set of cases and a considerable increase in the other. A similar striking contrast is seen in the treatment of these diseases; the one requiring the most active antiphlogistic remedies, the other being aggravated by them. Judging from the treatment, we conclude phrenitis to be inflammatory, and we consider delirium tremens as not in the smallest degree partaking of the nature of inflammation. Hence the excretion of the excess of phosphates may be regarded as resulting from inflammatory action going on in the brain, whilst the diminution of the same phosphates in delirium tremens must be considered as caused by the positive hindrance of that process of formation of phosphoric acid which in the healthy state is continually taking place.

Perhaps the following might be given, as at present the nearest approximation that can be made to the connection of these facts. That in the healthy state, a portion of the phosphoric acid which is excreted results from the action of the inspired oxygen on the phosphorus of the phosphorized fatty matter of the brain. In delirium tremens, either from the action of the substances which cause this state of disease, or from the peculiar state of the nervous structure, this production of phosphoric acid by the action of oxygen is almost entirely hindered; whilst in inflammation of the brain, it seems probable from these experiments that an increased formation of phosphoric acid results from the inflammation of the nervous structure. In other words, it seems necessary to admit that inflammatory action and the action of oxygen must be in some close relation to each other;—a relation which is also brought before us in the increase of temperature of inflamed parts, and in the peculiar oxides of albumen which form the chief constituents of the inflammatory crust of the blood. The admitted influence of the action of oxygen on life would lead us to expect that it would be an important element in disease.

The excess of action, or the want of action, of the so-called vital gas may, possibly, ultimately be proved by the balance, not only to be traceable by its effects on the nervous tissue, but other tissues may give a corresponding result. With this view the variations of the sulphates in disease deserve the most careful investigation.

AN ACCOUNT OF A CASE
OF
ENCEPHALOID DISEASE
OF
THE ENDOCARDIUM.

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THE subject of the following remarks was admitted, in the course of November 1846, into St. Bartholomew's Hospital, under the care of Dr. Roupell, to whom I am indebted for the permission to communicate the case to the Society, and whose attention in investigating all the symptoms is the best guarantee that the imperfections of the diagnosis which constitutes the chief interest of the case did not arise from any carelessness or inaccuracy.

William Pavey, aged 48, extremely weak and emaciated; a man of temperate and steady habits, married three times, having had one child by the second wife, but none by the third, whom he married twenty months ago, was under observation for about the last fortnight of his life. He had always had good health till two years ago, when his right testicle became enlarged, causing him great distress by its weight, and so incapacitating him for work that he submitted to its removal in the early part of August last. The scrotum had not ulcerated, and the wound healed readily after the operation, leaving a neat, firm cicatrix.

There was at this time no suspicion of the disease extend-

ing further than the testis, and the enlargement of this part was not supposed to be owing to encephaloid disease; but suspicion was aroused by the observation of a soft tumour in the epigastrium, which was noticed while the patient was recovering from the operation. However, in the course of a month he resumed his employment as a warehouseman, but gave it up again, in a few weeks, from sheer debility. A violent attack of diarrhoea about this time completed his prostration, and since then he kept his bed, till his death. He had no pain, but merely extreme weakness, with constipation and occasional vomiting, his food returning unchanged sometimes as much as ten hours after ingestion. He had never suffered from cough nor haemoptysis, nor anasarca, and there was no enlargement of the superficial veins of the abdomen.

To sustain his strength, and to allay the vomiting which had only existed within ten days previous to his admission, were the indications acted on; the latter with great success by means of hydrocyanic acid; the former to little purpose, for he died on Nov. 24, twelve days after admission.

The examination of the various organs during life elicited the following facts:—

Beneath the lower end of the left sterno-mastoid muscle was a tumour about as large as a chestnut, which was said to have been only lately noticed.

Respiratory murmur was almost absent, and there was some dullness on percussion beneath the left clavicle; the heart's sounds were loudly transmitted below both clavicles. A systolic murmur was audible at the apex of the heart, of a faintly musical character, and one of a soft blowing character was heard in both the pulmonary artery and the aorta.

In the epigastrium there was a soft tumour, slightly painful on pressure and doubtfully fluctuating, which was said to vary in size at different times. Below and a little to the right a gurgling sensation was communicated to the fingers on making light rapid pressure; below this, in the right umbilical and lumbar regions, was a large hard tumour, evidently behind the intestines, which gave a clear sound on percus-

sion everywhere except over the soft epigastric tumour: once only during life was the lumbar tumour dull on percussion.

The urine, the secretion of which had been temporarily suppressed, being examined on the earliest opportunity, was found to be loaded with urates and pus globules, in which last the nuclei were singularly distinct when viewed under the microscope. Let us compare these results with those of the examination of the body after death.

The brain was not examined. The cervical glands were generally enlarged, but apparently healthy, except one mass which lay behind and to the outer side of the left common carotid, and a soft swelling, apparently of a glandular origin, (being the tumour noticed during life,) lying behind the left sterno-mastoid, with the fibres of the scaleni muscles spread over it:—this mass extended on the outer side of the carotid artery, and above and somewhat behind the subclavian artery which crossed its lower end; and was itself traversed in front by the phrenic nerve. It had only loose attachments above and all round; but below, where it sank behind the clavicle, it was firmly attached to the costal, which adhered about the same place to the pulmonary, pleura. On removal, it appeared to be a firm elastic cyst, of about two inches diameter in the long, and one in the shorter, axis. On section, a dirty, purple grumous substance exuded, leaving a thin but strong membranous bag, whose inner wall was rough with adherent particles of the same grumous matter.

The apices of both lungs were adherent by firm old cellular bands; the rest of the left lung was also more or less adherent by looser fibrin, and the pulmonary pleura was in some points thickened. Throughout each apex were scattered numerous small grey-yellow granules, with some chalky nodules (apparently quiescent tubercles or their remains), and the lungs, to the same extent, were largely infiltrated with black pulmonary matter. Elsewhere they were healthy, except near the base of the right, where there were a few raised patches, of about three lines in diameter, and of a dull red colour with a somewhat lighter-coloured border: beneath these the

substance of the lung was softened to the depth of a line or more.

The heart was of about the natural size, externally healthy, with the exception of a pale spot on the anterior surface of the right ventricle: the walls were thin and flabby, the valves efficient, and all the cavities healthy except that of the right ventricle, which appeared nearly filled up by some roundish masses of a dull red colour and soft consistency, springing from a point corresponding to the pale spot seen on the external surface of the heart: their free rounded extremities were directed towards the pulmonary artery, and the tip of the longer lobule was not more than an inch distant from the lower edge of its valves. Parallel to this, and to its right, lay a similarly-shaped but shorter mass, which was separated from the next mass by the *carneæ columnæ* giving origin to the *chordæ tendineæ* for the left flap of the tricuspid valve; so that the next lobule, which was shorter than the two preceding, thicker and somewhat cleft at its free end, projected into the ring of the valves, and, though it might not have actually intervened between the edges of the flaps of the closed valve, it must have interfered with the play of the *cordæ tendineæ*. Below lay two or three roundish flattened masses extending quite to the apex of the ventricle.

Just about the attachment of the larger masses, the muscular substance of the heart appeared infiltrated with *encephaloid* matter; but nearer the apex, where the tumours were smaller, the muscular substance of the heart was comparatively, if not quite, free; the masses being attached by roots ramifying among the *carneæ columnæ*, and adhering to the surface, without actually penetrating into the muscular substance, of the organ.

In the usual situations existed small, rough, red, fibrinous coagula, to which, in colour, though not in consistency, the *encephaloid* masses bore a great resemblance.

The results of the external examination of the abdomen, after death, were much the same as already noticed during life, except that the tumour in the lumbar region could not be

so distinctly felt. On reflecting the integuments, which were free from all attachment to the tumour and very thin, the edge of the liver appeared from beneath the right costal cartilages, and was small but healthy; below this was the gall-bladder, containing nearly 4 oz. of dark bile. Next, on descending towards the pubes, there appeared a large round soft swelling (the epigastric tumour already mentioned), over the front of which the pyloric end of the stomach, the first two portions of the duodenum, and the pancreas with the superior mesenteric vein, were visible. The colon, corresponding in situation to the place where the gurgling had been felt during life, crossed the abdomen below these, turning up at a sharp angle from the right flank. Below this was a space, over which was spread the peritonæum from the back wall of the abdomen, and part of the mesentery, the small intestines having fallen away into the cavity of the pelvis and the hollow of the flanks on either side.

The tumour was now seen to be a large, imperfectly lobulated mass, of a soft elastic feel and dark purple colour, weighing about 5 lbs., and occupying chiefly the right lumbar region, but extending across the spine into the left also. It was free from all but ordinary cellular attachments to the spine; the left kidney, the pancreas and the duodenum, had contracted no unnatural adhesions to it; and the spleen, which was quite free, was, like the other viscera of the abdomen, perfectly healthy; the right kidney alone was diseased. This organ was connected very firmly with the tumour by old cellular tissue; it presented no traces of encephaloid disease, but the surface, beneath the capsule, was ecchymosed in points; in other points were yellow deposits of pus extending a short way into the cortical structure of the kidney,—the ordinary appearances of suppurative nephritis; and in one place there was a funnel-shaped depression, about four lines in diameter, communicating with the pelvis of the kidney which was filled with pus.

The tumour, on a closer examination, was found to be composed of a soft brain-like matter, closely resembling that in

the neck, readily exuding on the division and pressure of any of its large lobular masses. The aorta and vena cava ascendens ran through its substance, these two vessels being separated at the upper end of the tumour by a space of two inches. The coats of the artery were healthy, as were also those of the vein at its upper extremity; but the cavity of this vessel was almost obliterated below by loose yellow flocculent masses adhering to its walls: the left common iliac vein was obliterated by a firm laminated coagulum, the right at its entrance into the tumour was ulcerated; and its union with the left iliac, where this vessel had become pervious, to form the cava, was not distinguishable in the softened mass. The lumbar veins were much enlarged but healthy.

Numerous enlarged glands were scattered about in more or less close connection with this tumour. In the track of the right external iliac vein were a few small hard bodies; the corresponding parts on the left side were healthy.

The left testicle was small, and apparently healthy. The right, which Mr. Childs of Fore-street (who had removed it in August) has liberally deposited in the Museum of St. Bartholomew's Hospital, forms a large round ball, the enlargement being chiefly confined to the body of the organ. The cavity of the tunica vaginalis is obliterated by a thin flake of fibrin, adherent on both sides and completely circumscribing the morbid growth, which in one part has perforated the visceral layer of the tunica vaginalis, but is separated by the flake of fibrin from the reflected layer. The substance of the organ is irregularly lobulated, one large lobule having apparently cracked away from the rest, from the effects of the solution in which it has been kept; one or two small yellow spots are to be seen in it, but there is no appearance of softening of the organ.

On examination of the tumours under the microscope, with the assistance of Mr. Paget, that in the heart was found to be made up of a loose opake tissue and granular matter. Of the tissue itself, nothing could be made out satisfactorily; the tumour seemed for the most part composed of granules,

these being roundish bodies, pale, granular, but much smoother than pus globules, mingled with caudate corpuscles of varying form, and very exactly resembling the awned seeds of some grasses; there were also some cells with nuclei, which were granular, like their parent cells. There were many small flat globules and some crystals, but none of these last were found in the abdominal tumour, which was examined in the recent state. In the testicle, nothing could be clearly made out, the substance seemed to have been so altered by the action of the solution; all that was distinctly discernible being a few globules, such as constituted the mass of the tumours in the heart and abdomen.

The subject of encephaloid disease of the heart and testis has been so lately before the Society, that it seems best to confine the present communication to those parts of the subject on which the case under consideration appears to throw some light; namely, the diagnosis and the mode of origin of the cardiac disease: merely noticing by the way, with regard to the diagnosis of the abdominal affection, how the discovery of pus in the urine, which was so clearly explained after death, led to the rejection of the previously formed and correct opinion, that the lumbar glands were the seat of disease.

The state of the heart was not examined hastily, but more than once with the greatest care, on account of the suspicion that there was medullary disease of the pericardium,—the friction of the hair of the chest under the stethoscope having given rise for the first two days to this suspicion. From the facts elicited it does not appear that any murmur existed at the base of the heart characteristic of the presence of the tumour; for as there was also a murmur in the aorta, it would be unfair to conclude that the murmur in the pulmonary artery arose from the presence of the tumour in the right ventricle;—rather the murmur in both situations arose from a common cause, namely, the condition of the blood. But, considering the position of the tumour in relation to the tricuspid valve, it seems natural to refer the murmur at

the apex of the heart to imperfection of this valve, caused by the presence of the lobule already alluded to within the circle of the chordæ tendineæ ; though the absence of a more exact definition of the situation of the murmur than the general statement of its having been audible at the apex affords, is to be regretted in a case fitted as this was to test the accuracy of particular diagnosis between imperfections of the mitral and the tricuspid valves.

On the subject of the auscultatory signs of encephaloid diseases of the heart, the following brief notice possesses some interest :—in a man aged 45, suffering from cancerous disease of the pylorus, who died in St. Bartholomew's Hospital in the spring of 1846, it was noticed three days before death that the heart's sounds were not clear, though unaccompanied by any murmur : on dissection, it is recorded that there were found 2 oz. of blood-red serum in the pericardium, the membrane being highly injected, not coated with any fibrin, but studded, especially about the reflection of the pericardium from the heart to the great vessels, with raised, flattened, yellow bodies, which were about the size of melon seeds, and separate over the face of the ventricles, but larger and almost confluent about the base ; similar bodies existed also on the right pleura and in the right crus of the diaphragm. Probably auscultation might have detected a friction sound at a later period when pericarditis had commenced ; but certainly the encephaloid disease did not itself give any local signs of its presence, either physical or constitutional. Its existence in the pericardium would have been manifested only by the signs properly referrible to the inflammation which it produced, as in the interior of the heart only by the incidental circumstance of its interfering, as any other foreign body might do, with the heart's action.

It is impossible to read the collection of cases of cancer of the heart presented by Bouillaud (*Des Maladies du Cœur*, tom. ii. 428, 2^{me} ed.), or the more complete summary of Dr. Walshe (On Cancer, p. 368), and not still agree with Dr. Hope (On Diseases of the Heart, p. 355, 3rd ed.), that

sufficient materials for a history of cancer of the heart have not been collected. The cases just narrated, by showing the uncertainty of the diagnosis, only tend to confirm this remark, and the characteristic symptoms of suffering from this lesion are equally left in obscurity. Indeed, after eliminating the particulars which are common to other forms of disease of the muscular structure or valves of the heart, there are very few left that throw any light at all on the matter: for, to apply to the particular case before us what Dr. Latham remarks in a more general sense, "they have nothing to do with the essence of the tumour in question, and profit us nothing in suggesting any method of cure. They have, in fact, no rational treatment; and simply for this reason, because they have no essential symptoms."—(Latham on Diseases of the Heart, ii. p. 131.)

We proceed, lastly, to consider the mode in which the cancer had originated in the heart in this particular case. Looking at the history of the case, and the size of the abdominal tumour, it appears probable that the cardiac affection was secondary to the abdominal. And, taking the size of the tumours in the heart itself and the amount of the surrounding morbid changes again as our guide, it would seem that the tumours at the apex were more recent than those nearer the base of the heart. It might be argued with some plausibility from this case by itself, that, whatever the form and mode of connection of the tumours at the apex, such might be inferred to have been at the earliest period of their existence that of the tumours nearer the base, though it was now concealed in their further growth. But we prefer to investigate the matter by showing what light the published cases of endocardial cancer throw on one another.

Of the six cases of endocardial cancer which we are acquainted with, only four are applicable to the present purpose; the case reported by M. Cruveilhier from M. Payan, if indeed it be a case of cancer at all, and the second of Mr. Prescott Hewett's cases lately communicated to the Society, (Med. Gaz., vol. xxxviii. p. 889,) not exhibiting any very cha-

racteristic forms of the disease, and the last merely presenting a soft deposit which could be determined to be of a cancerous nature only by the history of the case and microscopic examination. The other four cases of primary disease of the endocardium,—primary as distinguished from cancerous growths, which have perforated the walls of the heart, whether originating in the muscular substance of the heart itself or in any neighbouring part—seem well to illustrate the different stages in the growth of these tumours.

The mode of arrangement presumes an analogy to exist, as suggested by Rokitansky, (Path. Anat., Bd. ii. s. 469,) between cancerous tumours of the endocardium, and the more commonly known globular vegetations of Laennec (Auscult. Mediate, tome iii. p. 344).

But as the analogy, owing to the essential difference between the component particles of the globular vegetations and cancerous matter, is only strictly applicable to the earliest stage, it will be sufficient to observe that these vegetations are supposed to have their origin in spontaneous coagulation of the blood, the result of abnormal conditions of that fluid; the coagula thus formed becoming adherent, by means of pedicles, to the valves or muscular walls of the heart, especially in the tips of the auricles or ventricles.

The cases seem to stand thus: First stands M. Cruveilhier's case, (Anat. Path., liv. 40, pl. 2,) which, considering its origin from the venous sinus, and not from the tip of the right auricle, and its proximity to the cancerous parotid tumour, is perhaps no more than primary venous cancer, (Rokitansky, Path. Anat., Bd. ii. s. 657,) which by the accidental circumstance of its position had been able to grow to so large a size, and to destroy life mechanically without losing its original, pedunculated, globular form. Next—or if the description be most strictly confined to the heart, these would illustrate the earliest form, for in no way are they the result of the further development of the form just noticed—come Dr. Sims' third case, (Med.-Chir. Trans., vol. xviii. p. 296,) and the tumours at the apex of the ventricle

in the case now under consideration. In Dr. Sims' case all the disease was, "in the appendix to the right auricle, a mass of adventitious deposit the size of a small walnut," which the context does not allow us to consider as anything but cancerous. The tumours at the apex in the present case, with their round form, and their sole connection by a narrow pedicle either with the endocardium or with the fibres of encephaloid matter which were interlaced with the carneæ columnæ, bore so close a resemblance to ordinary globular vegetations, that, but for the remainder of the growth, it might have been difficult to decide upon their exact nature.

Mr. Hewett's first case, as far as the brief notice in the *Medical Gazette* will allow me to infer, stands next as illustrating the mode in which these masses increase in volume, the same being also discernible in the tumours near the base of the heart in the present case; for here the analogy ceases between these growths and globular vegetations, a pendulous lobular mass supplying the place of the round body before seen, and infiltrated cancer of the muscular substance of the heart spreading from the widened base of the tumour which originated in the endocardium.

CASE
OF
INTERNAL STRANGULATION OF
INTESTINE
RELIEVED BY OPERATION.

BY GOLDING BIRD, A.M., M.D., F.R.S.,

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AND

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Received January 8th—Read February 9th, 1847.

THE following case will, I hope, be regarded as possessing some claims to the notice of the profession, as it affords a well-marked example of a class of cases as distressing and serious as any which can possibly fall under the care of the physician or surgeon. Recorded experience is almost barren in information regarding the results of attempts made to relieve insuperable constipation, when depending upon internal strangulation, by surgical interference. It is indeed a question in the minds of many, whether such interference is justifiable at all ; but I think this question could hardly be raised if our diagnosis of the nature and seat of destruction in such cases could be rendered more satisfactory. The following case will not be regarded by some as holding out very flattering prospects of success, inasmuch as it ended fatally ; still I do think that it is important, from its showing that a diagnosis of the nature and seat of obstruction is not always impossible : and I think that had the proposed operation been acceded to earlier, and not deferred until the strangulation

had continued fifteen days, the patient's recovery might have been fairly hoped for.

Dec. 21st, 1846.—I was called to Bocking in Essex, to meet Messrs. Harrison and Holmstead, of Braintree, in the case of Mr. C—, who was said to be labouring under insuperable constipation. I found a young gentleman, twenty years of age, of spare, but rather muscular frame, lying in bed on his left side, with his knees drawn up, and his countenance expressive of great anxiety. The face was flushed, the eyes bright and lustrous ; the surface cool ; pulse between 80 and 90, rather weak. His immediate history was as follows :—Eight days previously, Mr. C— was as well as usual, having merely had (as was common with him) the bowels constipated for a couple of days. In the morning, whilst in bed, he became sensible of a *slight dragging, or sense of giving way, on the right side of the abdomen*, about two inches from the umbilicus towards the spine of the ilium ; this sensation did not last long, and was soon replaced by a sense of soreness and tenderness. So little impression, however, was made on his mind by these symptoms, that they were only elicited by careful cross-examination. During the following six days nothing passed from the bowels excepting with the aid of a copious enema (nearly three quarts), injected by means of O'Beirne's tube, by which the large intestines appear to have been completely emptied of their contents. In spite of the most assiduous treatment of his medical attendants, all efforts failed to attain any further relief from the bowels. All purgatives given by the mouth, including croton oil and calomel, were quite inert, and a tobacco enema proved equally valueless. On the day preceding my visit, a small portion of faecal matter had, however, escaped from the anus. Three days previously, being five from the commencement of the attack, sickness commenced, everything taken into the stomach being rejected almost as soon as swallowed. Soon after, the vomited matters presented all the characters of the contents of the small intestines. On examining the abdomen, the patient did not complain of positive pain on pres-

sure in any part ; a mere sense of uneasiness and distress being produced by firm pressure on the spot where the dragging was first felt, to the right of the umbilicus, as well as on a corresponding spot to the left of that region. There was no appearance of hernial protrusion. The abdomen was flat and collapsed ; its walls thin and by no means irritable ; so that the whole region admitted of minute tactile examination. The cæcum and colon appeared to be empty, and no resonance was elicited by percussion over the track of either of these intestines. No tumour nor any other abnormal condition could be discovered. I fancied, however, that an abrupt ridge could be made out by careful palpation about two inches along a line drawn from the umbilicus to the anterior and superior spinous process of the right ilium. The patient denied having, throughout his whole illness, suffered anything amounting to acute pain ; the only thing approaching to this of which he complained, was from spasm, relieved almost immediately by eructations of air from the mouth. He had never suffered from hernia, nor ever had haemorrhage from the bowels ; and he had never been in any way exposed to the influence of lead. There was nothing in his pursuits (those of a private gentleman) which threw any light upon the etiology of the disease. As there could be no doubt of the existence of some mechanical obstruction to the passage of the contents of the intestine, I examined carefully the previous history of the patient, in the hope of discovering some clue to the nature of the obstruction. It appeared that when a child of about three years of age, he had been the subject of mesenteric disease, and had for some time suffered from a distended abdomen, from which he very slowly, and with difficulty, recovered. Some years afterwards he suffered from some ailment of which he did not give a very clear account, but which, from what he recollects of its symptoms, appears to have been peritonitis ; and about four years ago he had a similar illness, under which he laboured for three weeks. During the whole of his life, from the period of his having been the subject of mesenteric disease, he had always suffered

from habitual constipation, almost always having recourse to aperient drugs, and frequently to medical assistance. So far as he recollects, however, he had rarely permitted two days, and still less frequently three or four, to elapse without obtaining relief from the bowels. Such was the history of the case from which a diagnosis was to be drawn. The absence of hernia, as well as of any previous haemorrhage from the intestines, or of exposure to the influence of lead, the improbability of the presence of malignant disease, proved the non-existence of some of the most ordinary causes of insuperable constipation. Taking all the circumstances of the case into consideration, recollecting the dragging sensations experienced by the patient, and the previous existence of peritonitis, I ventured to give an opinion that the mechanical obstruction depended upon a knuckle of intestine becoming strangulated in some manner under a band of false membrane. There was of course more difficulty in determining the portion of intestine strangulated. The character of the vomited matters, and the empty state of the cæcum and colon, at once referred the seat of the obstruction to the small intestines. On making minute inquiries respecting the secretion of urine, we found it, notwithstanding the distressing vomiting, to exceed 30 ounces in twenty-four hours. It was hence obvious that the absorbing surface of the small intestines could not have been very considerably interfered with, and induced me to express my belief, that the seat of strangulation was not many inches from the ileo-cæcal valve. Coupling this with the seat of the previous dragging sensation, it was rendered highly probable that the stricture would be found in the right iliac region. This opinion, as well as the probably fatal termination of the case, was frankly communicated to the patient and his friends. In conversing with the patient (of whom I must take this opportunity to remark that I never met with a young man of greater calmness of manner and higher moral courage) I canvassed the chances offered by an operation. Having had a scientific education and possessing a general knowledge of anatomy, he fully appreciated his con-

dition, and talked as calmly of submitting to an operation as if it had merely concerned a third person. He was merely anxious, in deference to the wishes of his anxious relatives, to protract it to the latest possible period. As to medical treatment, I merely strictly interdicted all purgatives, and directed an enema, containing 3*j.* Tinct. Opii (which had been previously twice used with great benefit), should be repeated each night. Small quantities of bland nutriment were ordered to be frequently administered. I also suggested making a trial of metallic mercury, with the hope rather than the expectation that it might by possibility aid in disentangling the intestine. The patient was anxious to take it, and fourteen ounces by weight, warmed to 90°, were administered.

I was favoured with a report from Mr. Harrison twenty-four hours after my visit. From this it appeared that the patient had remained in but little suffering, and had ceased to vomit since taking the mercury. Two more doses of the metal were administered, so that about 2½ to 3 pounds were altogether swallowed. On the following day some little hope was excited by the passage of a small quantity of faeculent matter, but unmixed with mercury. No important change occurred until Friday afternoon, Dec. 25th, the fourth day from my visit, when vomiting again occurred with violence, with a return of all the distress I had found him labouring under.

On the following day, Dec. 26th, being again requested to go to Bocking, I met Messrs. Harrison and Holmstead at 10 P.M. We found the patient with a more anxious look, but still not suffering any amount of pain; his skin was soft and cool; pulse 90, small, weak, threadlike; tongue moist, white in the middle, red at the tip and edges, but scarcely furred. He had just vomited about a pint and a half of matter from the small intestines. The abdomen was rather more distended than at my first visit, but in all other respects the same, with the exception of the muscular parietes being very irritable, and contracting spasmodically in a curious manner after palpation, but without any pain to the patient.

The question of the propriety of an operation was once

more entertained ; the patient completely understanding that it would merely give him a chance in favour of recovery, as I did not think it justifiable to hold out much hope of success from an operation concerning which we have really no experience. On taking my leave, I begged, in the event of the operation being acceded to, that care should be taken to provide a room heated to 88° or 90° .

Mr. C—— remained much in the same condition until Monday, Dec. 28th, when I received another summons to Bocking, and to take with me a surgeon, with a view to the performance of an operation. I therefore requested the assistance of my friend and colleague, Mr. Hilton, and we arrived at the patient's house at 9 o'clock p.m., just fifteen days from the commencement of the illness.

The patient was comparatively cheerful and very calm ; the abdomen was scarcely more distended than on my first visit, excepting on the left side, which was more prominent than the right ; the muscles were much more irritable, easily assuming a state of spasmodic contraction on the slightest manipulation. No considerable amount of pain or even uneasiness was experienced on pressing the abdomen. Pulse 90 ; skin soft and cool ; tongue moist, scarcely furred. The patient fully understood his perilous condition ; and it was with the full understanding of this on the part of his friends, and with the concurrence of his local medical attendants, that I gave my sanction to the operation. A room had been most admirably arranged by Messrs. Harrison and Holmstead, by heating it by means of two of Dr. Arnott's stoves ; all unpleasant dryness of the air being removed by allowing a small current of steam to enter the room. By these precautions, a steady temperature of 88° to 90° was maintained in the apartment into which the patient was conveyed. The further account of the case will be given by Mr. Hilton.

In the evening of Dec. 28, I accompanied Dr. Bird to see the case which he has just described. I found the patient presenting the symptoms of some obstruction to

the intestinal canal, and after a careful interrogation and minute external examination, I could arrive at no other conclusion than that which had been already expressed by Dr. Bird and Messrs. Harrison and Holmstead. Before commencing the operation, I explained to my professional colleagues the whole of the intended abdominal section and exploration, and the means to be employed to detect the position of the stricture, referring especially to our using the completely empty small intestines as our guide to the position of the obstruction. Some laudanum was given to the patient about half an hour before his removal to the room in which the operation took place.

The patient being placed recumbent on a narrow French bed, and the urinary bladder emptied by the introduction of a catheter, an incision was made in the median line of the abdominal parietes, extending from the umbilicus downwards to within an inch of the pubic symphysis, through the skin and areolar tissue. The edges of the divided skin became widely separated, to the extent of at least an inch, by which the linea alba and adjacent cellular tissue were exposed. A small incision was then carefully made through the linea alba tendon, immediately below the umbilicus, so as to perforate the peritoneum, without wounding the intestine. A small quantity, about 3*ij.*, of serous fluid coloured with blood escaped through the wound. A broad flat director was then introduced into the peritoneum, and so directed downwards, in very close contact with the abdominal wall, as to allow the lower portion of the median tendons of the abdominal muscles to be divided upon it with a scalpel. In making this incision, the edge of the left rectus abdominis muscle was exposed, and a small branch of an artery was divided upon which a ligature was placed: only a very small quantity of blood, gathered by sponging, was lost by the patient: a little more bloody serum flowed from the abdomen at the lower part of the incision. Several convolutions of small intestine, rounded, much distended and their parietes congested to a light claret colour, presented themselves at the opening, pressing for-

wards, and urging their escape from the abdomen. No solid effusion of lymph was noticed upon the intestines. I passed my hand into the abdomen, towards the right side, in the direction of the seat of the first painful dragging sensation experienced by the patient, that I might ascertain whether there were any adhesions to the abdominal parietes: not anything unusual could be detected by this examination, nor did the same kind of manipulation discover anything wrong on the left side. The distended intestines so completely blocked up the opening already made, that it was impossible to extend the exploration without enlarging the abdominal section: this was done from below upwards, upon the broad director, to about an inch and a half above the umbilicus, passing to its left side. More space having been thus obtained, the convolutions of the intestines were now somewhat separated from each other, with as little manipulation as possible and with all due gentleness, first on the right side, without noticing any cause of obstruction, or any empty intestine. The same kind of examination was then made towards the left side; and in the left iliac fossa, over the sigmoid colon, two portions of small intestine were seen firmly adherent to each other by a band of old solid cellular tissue, about half an inch wide and about an inch in length; this band appeared to be constricting very much one of the portions of the intestine thus held together. It was divided with a scalpel, and the two portions of intestine were liberated, but the duplicature seemed to be firmly held in the pelvis; no force was used to raise it. Tortion was used to a very small bleeding artery divided in this band, but not arresting the blood satisfactorily, a fine ligature was applied upon it, and subsequently cut short at the knot. I expressed my decided belief that this adhesion had not been the cause of the patient's symptoms. On turning aside this convolution of intestine, I recognized the sigmoid flexure of the colon, deeply placed, of a light, healthy colour, very small and to all appearance quite empty. I then determined to make a complete exploration towards the right iliac fossa. To do this, it was necessary to displace the

distended ilium, and to raise the abdominal parietes. A portion of ilium, about six or seven inches long and more fixed than the surrounding convolutions, was then seen, presenting the appearance of strangulated intestine, being of a darker colour and less elastic than the adjacent coils. This portion of intestine being elevated and brought forwards, the cæcum was exposed, collapsed, empty and very small, with its appendix contorted, and fixed by old adhesions; a portion of small intestine, exceedingly contracted, not bigger than the end of the little finger, and quite pale in colour, was at the same time brought into view. It was now obvious we were near the cause of obstruction, having distended and empty small intestine close to each other. After a careful examination of the parts, I ascertained that the six or seven inches of darker ilium, before referred to, had passed through an annular opening, formed in part by another portion of the same small intestine, and in part by some old membranous adhesions to the brim of the pelvis over the external iliac blood-vessels. I endeavoured, by well-directed pressure, to return the distended intestine through the opening, but could not accomplish my intention, although the intestine became somewhat reduced in size. Gentle traction was then made on the strangulated intestine at the opposite side of the opening through which it had passed, and after a little perseverance I succeeded in liberating it from its incarcerated position, and immediately the contracted and empty portion of ilium, between the constriction and the cæcum, began to be distended, indicating clearly the removal of the obstructing cause. The intestines were now replaced, but with some difficulty, in the abdomen. A continued suture was used to approximate the edges of the wound from above to below. A thin pad of lint was placed on the wound; and over it a few straps of strong adhesive plaster, about an inch and a half wide, were extended transversely from one lumbar region to the other.

During the operation, which lasted about an hour from beginning to end, the patient made but little complaint of pain, and did not feel either faint or sick, though he had one or two

slight hiccoughs: after the operation he was somewhat collapsed, perspiring, and having a pulse of more than 100 in a minute, small and feeble: there was no marked anxiety of countenance. The exposed intestines were kept warm by sponges, as well as by the high temperature of the room: the great omentum was not seen during the operation, nor were any recent adhesions of the intestines, to each other or to the abdominal parieties, visible.

About an hour afterwards, Dr. Bird saw the patient, and reported him as quite comfortable; and, not wishing him to be disturbed a second time, I did not see him again. Forty drops of Battley's solution of opium were given to the patient soon after the operation; he was then left in the care of his medical attendant, who reports as follows:—

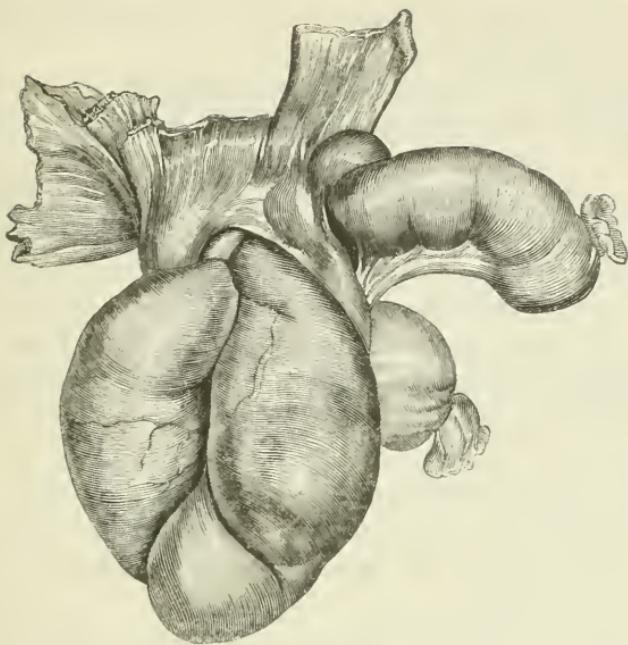
“When I saw Mr. C——, at twenty minutes past one on the Tuesday morning, he had rallied from the effects of the operation very much; he was calm and tranquil, said he felt very hot, and that his abdomen was very stiff, but he did not complain of pain, and there was neither vomiting nor hiccough; desiring to pass his urine, he did so in the bed. I gave him Cal. gr. iii. and Opii. gr. i. and a little water—he was quiet for half an hour; he continued very still and quiet till four o'clock, taking a little ice occasionally, to relieve his thirst. He was frequently still for twenty or thirty minutes, and I thought he had been asleep, but he assured me that he had not lost himself. The pulse was small, about 120: he had a second dose of Calomel and Opium at four. About five o'clock he had some conversation with his mother and sister, and gave them very explicit directions how to arrange the stoves, to keep up the temperature of the room: soon afterwards, he complained of dizziness and a sense of burning heat in his head, and at half-past five he became restless, and then delirious, calling out to be raised, or pulled out straight, throwing his arms about, and making efforts to get out of bed. A person on each side held his hands and soothed him: he was again quiet about half-past six. Very soon after that time, a death-like change came over his countenance, and it was evident that

he was dying. At a quarter to seven he was in *articulo mortis*, though he lasted till twenty minutes past seven. There was not any opportunity of giving brandy, for almost immediately after the delirious excitement subsided, the above-mentioned change took place, when he was no longer able to swallow."

Dec. 30th.—I examined the body at nine, p. m., about thirty-eight hours after death, with the assistance of Messrs. Harrison and Holmstead.

On removing the plaster and lint from the abdomen, which presented but little distention, all the part covered by the lint, including the wound and the adjacent skin for about an inch on each side, looked nearly white, and the rest of the skin in contact with the plaster was of a very dark hue, probably the result of the lead in the plaster having been decomposed by the sulphuretted hydrogen of putrefaction: a small quantity of bloody fluid had oozed from the wound at its lower angle. The suture retaining the edges of the wound in apposition being divided, a feeble adhesion between them, by recently-effused lymph, was noticed, and the same condition existed, by which some of the convolutions of the intestines were united to each other and to the abdominal walls. By elongating upwards the incision made during life, and by making an additional transverse cut through the abdominal walls from one lumbar region to the other, the convolutions of the intestines were exposed. They presented nothing remarkable in their grouping, all appearing to have found their right places, although they had been so much disturbed at the operation. The small intestines looked much less red than during life; indeed, at some parts they were rather pale. The cæcum was projecting and much distended, and the same distended condition existed along the whole length of the colon. The omentum was lying upon the intestines, just above the umbilicus: the liver was closely fastened to the diaphragm and abdominal parietes, by very old cellular adhesions. On attempting (for the purpose of examination) freely to displace the convolutions of the small intestines from each other, numerous old cellular adhesions were found

amongst them, causing some difficulty in tracing the continuity of the intestinal canal. The stomach and duodenum were both empty: neither of these parts presented any evidence of inflammation. The parietes of the jejunum were more vascular than natural, and the same may be said of the whole of the ilium above the strictured portion. Continuing to trace the ilium towards the cæcum, we arrived at that part where the band had been divided at the operation; and about two inches lower down there existed, between the same two convolutions, another strong cellular adhesion; and about three inches below this second band, occupying the lowest curve of the intestine and resting deep in the pelvis, was found the mercury, which had been administered at intervals, and which had not reached within two or three feet of the obstruction. Proceeding to follow the intestinal canal, we discovered about seven inches of ilium, somewhat darkly discoloured, larger than the adjacent intestine, not uniform in configuration, with some shreds of lymph upon it, and bounded at each end by a well-marked constriction; this was obviously the strictured portion of intestine which had been released from its fixed position at the operation, and close by it was recognized the abnormal opening, the cause of the obstruction, formed by almost a circle of small intestine (nearer to the large intestine than the part which had been incarcerated), maintained in that form by old adhesions, bounding, and so completing an annular opening, less than one inch in diameter, and fixed by other adhesions to the brim of the pelvis, close over the external iliac blood-vessels: through this opening, the subsequently strangulated intestine had passed, and through it likewise it had been repassed by traction, at the operation. This abnormal opening was about fourteen inches from the lower termination of the ilium. All this portion of the ilium, between the seat of constriction and the cæcum, was exceedingly contorted and very much fixed to the surrounding parts by strong adhesions.



A view, from the right side, of the strangulated intestine, which has been drawn forwards, so as to expose the boundary of the opening through which it had passed.

It was very satisfactory to notice that there were not any local indication of bruises or ecchymoses on the intestines.

The cæcum was distended by pultaceous faecal matter, well coloured with bile—the same distended condition by faecal matter was found through the whole length of the colon to the upper part of the rectum. Some of the mesenteric glands were much enlarged.

The kidneys, liver and spleen were examined by numerous incisions being made into them: they did not present anything worthy of remark, in reference to the immediate cause of death, except their pale condition: the mucous membrane of the lower parts of the jejunum and ilium was excessively congested, especially at the edges of the valvulae committentes—not any exudation of blood had occurred, nor was any ulceration of the mucous membrane detected in the portions of intestine examined. About half an ounce of

serum highly-coloured with blood was found in the pelvis, but no clot of blood.

The urinary bladder was nearly empty.

The heart was small, flabby, pale, and almost emptied of blood; but this latter condition may be in part attributed to the pressure to which the heart was subjected in removing it from the thorax by an opening through the diaphragm; all the valves were healthy; the two coronary arteries arose close to each other, above one of the aortic semilunar valves; there were no adhesions of the heart to the pericardium. Both lungs were sound; there were some few old pleuritic adhesions towards the base of the right lung.

Having thus detailed, without comment, all the essential circumstances of the surgical portion of this interesting and, I think, important case, it is necessary I should append one or two observations.

On visiting the patient I had no difficulty in agreeing with the opinion already entertained and expressed by Dr. Bird, as to the cause of his symptoms, and the probable seat of that cause; and knowing from my own experience in many similar (or, at least, believed to be similar) cases, the fatal issue which was then immediately impending unless some additional medical or surgical aid could be obtained, I did not hesitate to acquiesce in the suggestion of an operation, nor did I hesitate to undertake what, after due consideration, I felt I was justified in recommending, although quite alive to the extreme danger of such a proceeding, and, at the same time, not unmindful of the associated responsibilities of acting without the authority of precedent, and against the strongly-urged opinions and arguments of some surgeons in relation to two points in the case—viz. the possible failure in any attempts to find the obstruction, and even if found, the probable inability to relieve the constriction. These two latter reflections, not being in accordance with the conclusions at which I had arrived by *post-mortem* examinations in like cases, did not contribute, in my mind, any

urgent objection to the propriety of the trial ; nor did they portend, in my opinion, any insurmountable difficulty : and I think the circumstances connected with the progress of the operation in this case substantiated the correctness of such a conclusion.

I regard the direct results of the operation as very satisfactory : the hiccough subsided—the vomiting ceased—the intestinal obstruction was relieved—the contents of the small intestines began to pass towards the large intestines, immediately the incarcerated portion was liberated and replaced in its natural position, as evidenced by the distention and vermicular motion observed in that part of the ilium situated between the constriction and the cæcum, which portion had been previously noticed perfectly contracted and quiescent.

At the *post-mortem* examination, the small intestines were nearly empty, and the large intestines filled with faecal matter as far as the upper part of the rectum ; indeed, I have little doubt, if this patient had survived another hour or may be a shorter period, that his bowels would have been most freely relieved. The *post-mortem* examination demonstrates the complete inutility of the mercury in this instance, as a mechanical agent, in overcoming the obstruction. It was administered at intervals, during life, and nearly the whole of it was found collected at the most depending parts of two adherent convolutions, far removed from the true seat of the obstruction, and with no impediment to its progress except that of gravitation.

The class of cases to which the subject of this communication belongs is well known, and is acknowledged by the Profession as offering very considerable difficulty in diagnosis, and, when satisfactorily recognised, as necessarily fatal if not relieved by mechanical assistance ; and such must inevitably have been the result in this instance had not the operation been resorted to ; for although the obstructed intestine was in my hand, I could not, by safe pressure, return it to its natural position ; but was compelled, as stated, to

use traction on the stomach side of the stricture, indicating pretty clearly, that not any other means, except that employed, could have been of any material service or aid.

Looking at the case in all its aspects, I think it offered as much obscurity, in most respects, as any one of its kind can present; nevertheless, there was no very great difficulty in discovering the seat of the disease, nor in acquiring the suggestions as to the method of reduction—viz. by traction.

Although we may perhaps, oftentimes, as in the case before the Society, judge by the patient's sensations and other concomitant circumstances or symptoms, of the probable position of the obstruction, it must be admitted that experience has not yet pointed out with sufficient certainty the extent of reliance which ought to be placed in the indications as to its precise locality: so long as any doubt remains in reference to the whereabouts in the abdomen, we are to look for the obstructing cause, I think it must be the safer plan to adopt the median section of the abdominal parietes; the section in the linea alba may be made amply sufficient (at least so I conclude from all I have seen) to examine, with due care, every necessary part of the abdomen; it has the advantage of not implicating muscular fibres, which would retract and leave such portions of the abdominal parietes very thin, and so induce a constant disposition to ventral hernia; and further, it avoids the risk of haemorrhage from wounding any considerable artery or vein, except there should happen to be an abnormal distribution of blood-vessels in the course of the incision.

In cases of external hernia, an early operation contributes much towards a successful issue; and we may fairly suppose a corresponding advantage might have been obtained had this operation been performed soon after the obstruction occurred, and before the intestines had become so excessively congested or inflamed, and before the patient's powers of constitutional reaction had been so much reduced by medicine, by vomiting, and by defective nutrition.

Reflecting on all the concurrent circumstances of this case, I cannot but conclude that they fully justify the proceeding which was adopted; and, I may add, encouraged by this case, notwithstanding its fatal termination, I shall not doubt the propriety of advising and pursuing the same plan in any other similar case, provided the indications be as clearly expressed. I believe this to be the first recorded instance of any surgeon in this country having succeeded in his attempts to relieve an internal strangulation of intestine, by such an operation as was completed in this patient; I trust it will be considered by the Profession, as a step in the right direction, and as an additional resource in our art, which may be conscientiously and honestly employed with the intention of prolonging human life.



CASE
OF
ELEPHANTIASIS.

BY GEORGE SOUTHAM,

DISPENSARY SURGEON TO THE MANCHESTER ROYAL INFIRMARY,
LATE SURGEON TO THE SALFORD ROYAL HOSPITAL AND DISPENSARY.

COMMUNICATED BY T. B. CURLING.

Received January 11th—Read February 23rd, 1847.

THE following particulars of an extraordinary enlargement of the right inferior extremity will, I trust, be considered of sufficient interest to merit the attention of the Fellows of the Royal Medico-Chirurgical Society.

The patient, an unmarried female of dark complexion and phlegmatic temperament, first came under my notice in the autumn of 1843. The disease had then existed about twenty years, and commenced when she was in her eighteenth year. It began on the dorsum of the foot, having been preceded by several attacks of deep-seated pain in the part, attended with febrile symptoms. After the œdema appeared it became permanent, gradually but slowly extending up the leg. With the exception of an occasional return of the fever and pain, which she described as having been of a dull throbbing character and always followed by an increase in the swelling, she felt very little inconvenience from the disease for the first eight years, beyond what was occasioned by its bulk. As it extended along the thigh, however, the pain be-

came more severe, especially in the groin; and the integuments, which had hitherto preserved their natural colour, began to be the seat of frequent erysipelatous attacks. These, besides increasing the tumefaction, were attended by the oozing of a clear watery fluid from the inflamed skin, and by the formation of an incrustation on some parts of its surface. A few years ago a large ulcer formed on the inside of the thigh, and recently three others appeared near the ankle. Although a quantity of watery fluid mixed with pus has been regularly discharged from them, there has not been any diminution in the size of the limb. The pain formerly occurred only at intervals, and was generally relieved by leeches and fomentations; but within the last few years, it has become so constant that she has been compelled to resort to opiates for relief.

During childhood, and up to the period of the commencement of the complaint, her constitution was unimpaired, and she had a healthy appearance until she began taking opium regularly. Since then, she has lost flesh and the countenance has become sallow. Before coming to Salford, she had from birth resided at Bolton, about nine miles from her present residence. Her family, though of humble extraction, are remarkably healthy, and the only cause assigned for the disease is the sudden cessation of the catamenia from cold, since which they have never re-appeared.

The drawing, which was taken in 1845, is a representation of the disorder as it appeared for the last four years of the patient's life. During this period, probably in consequence of the existence of the ulcers, there was no apparent alteration in the dimensions of the limb. A cast indicating its size and singular external conformation may be seen in the Museum of the Royal College of Surgeons. The measurement round the calf of the leg was 2 feet 9 inches, above the knee 3 feet 4 inches, and at the upper part of the thigh, including the nates, 5 feet 6 inches. The integument on the upper surface above the knee was not altered in character, but from having yielded in some parts more than in others,

the leg had a somewhat lobulated form. Pressure did not leave the slightest indentation, nor was it in the least painful when touched, excepting below the knee and on the inside of the thigh, the situation of the former erysipelatous attacks. Here the skin was inflamed and continually moist. The fluid, which with a lens might be seen exuding from the sudoriferous ducts, had a slightly acid re-action, and under the microscope was found to consist principally of water, containing a few fat globules and granules, crystals of the chloride of sodium and epithelium-scales. Round the edges of the inflamed parts a thick incrustation was formed, which appeared to be caused by the drying up of the secretion, as it could be easily removed, leaving the skin underneath sound. The sole of the foot was the only part of the limb not implicated in the disease.

Her general health afforded no indication of having suffered. There was nothing, at any time, unusual in her appetite; bowels occasionally constipated; urine scanty and high-coloured, depositing a sediment which, when examined microscopically, generally consisted of crystals of urate of ammonia.

Though prevented from taking out-door exercise, she was able to go about the house and assist in the ordinary household duties, until within a few weeks of her death, which took place on the 21st of November last from an attack of dysentery. During the last few days of her life, there was considerable increase in the urinary secretion, sometimes amounting to eight pints in the twenty-four hours, and the discharge from the limb was much greater than on any former occasion.

On examining the body twenty-six hours after death, the enlargement was found to have been caused by the deposit of a dense white lardaceous substance, interspersed with fat, in the subcutaneous cellular tissue. When cut into layers, a small quantity of sero-sanguinolent fluid oozed out, and a few small vessels might be seen traversing its structure; but beyond their presence it did not present

any marks of vascularity. The cellular tissue beneath the superficial fascia was not at all affected. The muscles, though smaller than usual, preserved their natural appearance and situations. There was no enlargement of the bones or disease in the joints. The principal venous trunks were much larger than natural, distended like injected arteries and, when divided transversely, were patulous. Their external coat was thickened, and, except in a few places, the middle and internal one could not be traced, both having apparently been converted into a thick fibrous substance, disposed round the vessel in laminæ, not unlike what are observed in aneurismal tumours. The outer ones were of firm texture, of a pale brown colour, and connected to the external coat by fibrous bands. Those nearer the centre of the canal were soft, spongy, and had several flocculi on their surface. The same appearances existed in all the smaller veins in the diseased structure, which, when divided transversely, resembled arteries filled with coagula. Though considerable obstruction was offered to the passage of the blood through the principal veins, they were not completely obliterated in any part of their course. Several of the smaller ones, however, were impervious. The saphena was converted into a thick fibrous cord, and a large organized coagulum was found at its connection with the femoral vein. The arteries were small, and their coats thin. The nerves presented nothing unnatural, and the glands in the groin could not be traced.

On opening the abdomen, the diseased state of the veins was found not to have extended beyond the groin; those of the pelvic cavity were healthy, and the uterine organs sound, excepting that a small cyst, about the size of a walnut, was attached to the left ovary. The liver was small but normal. The spleen was slightly enlarged, and in a complete state of hepatization. The heart, lungs and kidneys were healthy. The lardaceous substance was submitted to the action of boiling water for upwards of sixteen hours, but yielded no gelatine. A portion, after being boiled in ether which ex-

tracted numerous fat globules, presented a cellular structure under the microscope. Another portion, that had not been acted upon by any chemical re-agent, appeared, with a magnifying power of 400 diameters, to consist of nucleated fibres, epithelium-scales, fat globules and granules. The skin was also carefully examined, but beyond slight hypertrophy of the epidermis and cutis nothing unusual was detected.

Though cases occasionally occur in this country resembling in a mild degree the Arabian elephantiasis, I am informed by several medical friends who saw my patient, and who had witnessed the disease amongst the natives of South America and the West Indian islands, that even there the tumefaction seldom attains so great a size as was observed in the case now related.

The *post-mortem* examination tends to show that the disease commenced in the veins, being probably the result of repeated attacks of inflammation of the capillaries, which gave rise to the pain and febrile symptoms in the earlier stages of the complaint. From the local nature of the pathological changes, the deposit found in the veins would appear not to have been formed immediately from the blood, but from the effusion and consolidation of lymph in their coats, the coagulation of the blood in the folds of the flocculi contributing to fill up the calibre of the vessels.

Several authors on elephantiasis allude to a morbid condition of the veins; and Dr. Wise* has written an excellent paper to point out that it is produced by inflammation of them. He says, "When the groin and thigh are examined in the acute stage, a swelling may be discovered, which extends to the knee-joint, or even to the foot, following in this course the various branches, and is indicated by the

* Transactions of the Medical and Physical Society of Calcutta, vol. viii. 1835.

pain and sensibility of the part on pressure: the neighbouring cellular tissue of the thigh and leg is swelled, over which a net-work of veins frequently appears, having, in some places, a hard, round, cord-like feel, with an erysipelatous blush of inflammation over the course of the veins thus affected. The state of these vessels are best observed in the acute primary attacks of the disease. In one case, I found, after a paroxysm, the trunk of the saphena major vein prominent on the bent knee-joint, with a hard cord-like feel, and a swelling upon the anterior part of the leg, from which many branches of the vein affected, during the paroxysm, had arisen."—P. 159.

He further observes, "I have endeavoured to avail myself of opportunities of examining elephantiasis in its different stages, but especially in its primary invasion, and am confirmed in my opinion that elephantiasis is produced by an inflammation of veins. When so affected, a consolidation of blood takes place in the trunks, which extends to the smaller branches and impedes the circulation in the part."—P. 178.

Dr. Towne* also remarks that the veins in the vicinity of the inflamed part during the acute attack "are found much distended with varicose swellings, which are very apparent from the knee down to the extremity of the toes."

It may, therefore, be inferred that the disease is the consequence of phlebitis, which, though usually regarded as being prone to terminate in suppuration, (probably from its frequent occurrence as a secondary disease,) where it exists as a primary affection, or the predisposition to it has not been induced by causes having a tendency to assume a typhoid or purulent character, is generally arrested at the adhesive stage.

Occasionally, as in the present case, the venous trunks

* Treatise on Diseases the most frequent in the West Indies. Lond. 1726.

afford no indication of being affected in the earlier stages,* the inflammation apparently commencing in the capillaries, and remaining for some time limited to them; consequently, if, under such circumstances, the limb be not carefully examined, the true nature of the complaint may escape detection. It is, probably, on this account, and the limited opportunities which pathologists have had of dissecting the parts affected, that so much discrepancy exists respecting its origin.†

The conflicting opinions respecting phlegmasia alba dolens may also admit of a similar explanation; the disease originating in the venous capillaries in those instances where no abnormal appearances have been found in the femoral or iliac veins, and the previously existing derangement of the system during the puerperal state having led to its development, as wounds cause phlebitis in parts remote from the seat of injury: where the femoral or iliac veins have been obliterated without any distinct manifestation of the complaint having extended from the uterus, the capillaries or even the larger vessels themselves may have been the part of the venous system primarily attacked; the affection assuming the acute form, puerperal phlebitis, or the chronic, phlegmasia alba dolens, according to the septic power of the causes which have given rise to it.

The albuminous substance sometimes met with in the cellular tissue of infants, presents characters somewhat analogous to what is observed in elephantiasis; the congested state of the veins which usually accompanies it showing, that in all probability it arises from some morbid alteration in them.

It would appear, therefore, that the immediate cause and

* In other cases, the local inflammation is confined to the foot and leg, which become swelled, hot, and pit on pressure; while the lymphatic vessels and glands of the thigh and groin continue without any apparent change.—Wise, *l. c.*, page 159.

† Chevalier, Med.-Chir. Transactions, vol. ii. 1811. Alard, Observations (Nouvelles) sur l'Elephantiasis des Arabes. Paris, 1811.

pathological changes of elephantiasis bear an intimate relation to those of phlegmasia dolens, and the induration of the cellular tissue in new-born children ; the apparent differences depending on the degree of venous obstruction and on the remote influences which have originated it.

EXPLANATION OF PLATE I.

This plate represents the appearance exhibited by the diseased limb in Mr. Southam's patient, during the last four years of her life.

PLATE 1.





ON
TUBERCULAR PERICARDITIS,
WITH PATHOLOGICAL AND PRACTICAL REMARKS.

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It may appear to many members of this Society almost a work of supererogation to describe another variety of pericarditis, after the extended inquiry into the causes of that disease by Dr. Taylor, which was published in the 28th volume of our Transactions. Considering how much attention has been paid to diseases of the heart during the past twenty years, their history might indeed by most persons be regarded as nearly complete, and such an opinion has been expressed upon several occasions, and was so by M. Andral, when he published, in 1829, the 1st volume of the *Clinique Médicale*. That able pathologist commences his clinical history of cardiac diseases with the following remarks:—"Since the publication of the immortal researches of Corvisart, and of numerous other works subsequent to his which have still further enlarged the dominion of science, the history of diseases of the heart and its membranes ought to be regarded as almost complete." Notwithstanding this prediction, how much has our knowledge on this subject been extended since the publication of Andral's valuable work, by the labours of Hope, Bouillaud, Bertin, Latham, Watson, Taylor, and others.

Dr. Taylor, in his essay on the causes of pericarditis (*loc. cit.*), has enumerated, and I think correctly, the three following as the most influential causes of that insidious disorder; —(1) Rheumatism; (2) Granular degeneration or Bright's disease of the kidney; and (3) the extension of inflammation from contiguous tissues to the pericardium. Although Dr. Taylor states that he believes other causes operate in the production of the disease, he does not intimate that he is acquainted with the variety which I am about to describe as Tubercular Pericarditis.

In a review of the causes of pericarditis as they are detailed by different writers, Dr. Taylor has not quoted any one who describes tubercular granulation of the pericardium as the exciting cause of inflammation of that membrane. Dr. Latham, in his elegant and instructive "Lectures on Diseases of the Heart," only incidentally alludes to this variety of the disease of the pericardium; although in the course of that physician's large hospital experience, it is most probable that some cases of this insidious and latent disorder must have occurred, but passed by unnoticed. Dr. Latham states that his experience of pericarditis is, like that of other physicians, mainly derived from what it is as an accompaniment of acute rheumatism. He remarks that he has seen the disease under other circumstances, but it has been very seldom; so seldom indeed, that he has little acquaintance with other conditions, external or internal, conducing to it. He can neither tell whence to look for it nor when to expect it, except when it occurs as a part of acute rheumatism (vol. i. p. 136). This author also remarks, that the pericardium is not exempt from those diseases which result in formations different from the natural structures of the body, such as tubercle, carcinoma, &c. But these diseases have not any special pathology respective to their seat within the heart, nor any distinctive history, diagnosis or treatment (Lect. 24, vol. ii. p. 120). It is hoped that the present communication may extend our knowledge on this subject, and point out other conditions of the system when pericarditis may be expected to occur.

The existence of tubercular deposits on the pericardium, both on its serous and fibrous surfaces, has been known to the morbid anatomist long anterior to the possession of any method of detecting pericarditis in the living person.

Dr. Baillie has recorded an instance of tubercles in the pericardium, and remarks that this disease is very rare there, and "could not have been guessed at in the living body." (Morbid Anatomy, p. 9.)

Laennec, in his celebrated treatise (vol. ii. p. 658), states that he had met with two cases of the development of tubercle in the false membrane of pericarditis; and which he thought had caused the acute to pass on to chronic pericarditis.

M. Louis reports that in 112 fatal cases of phthisis he had found the pericardium covered with false membranes three times, thus exhibiting evidences of pericarditis occurring in the course of phthisis, but he there makes no allusion to the presence of tubercles in the false membranes. In this author's Essay on Pericarditis, he relates a case of grey tubercular granulations of the pericardium, which he considered had acted as the exciting cause of the pericarditis. Andral has recorded two cases of chronic pericarditis (Clin. Med., vol. i. Obs. ix. & x.), which, from the details, may be regarded as examples of tubercular pericarditis; although it does not appear that that physician considered the inflammation of the pericardium as the consequence of the deposit of tubercles which were discovered in the fibrinous exudation. Dr. Taylor has concisely related (*loc. cit.*, Case 6, p. 457) the particulars of a case of pericarditis, the cause of which he could not assign, and which, if contrasted with those about to be related, will, I think, appear to have been an instance of tubercular pericarditis.

Rokitansky (Handbuch der speciellen pathologischen Anatomic, Band i., p. 378-9) has also described very accurately the various conditions of the pericardium when tubercle is deposited on that membrane; but he is of opinion that the tubercles result from the transformation of the fibrinous matter exuded during prior attacks of pericarditis, and not

that this inflammation is caused by the previous deposit of tubercle within the sac of the serous membrane. I shall again advert to this opinion of Rokitansky, which I believe is an erroneous explanation of the pathological phenomena.

This brief retrospect of our present knowledge of tubercular disease of the pericardium will render the following cases more intelligible and instructive.

Case 1.—Dominico Casa, an Italian musician, ætat. 24, was admitted, under my care, into St. Bartholomew's Hospital on Jan. 4th, 1844. His aspect was healthy ; he complained of some pain in his right side and shoulder, of general weakness and slight cough. His respiration was frequent though not laborious. His pulse was excessively feeble and irregular, occasionally intermitting. The sputa were at times slightly tinged with blood. Upon auscultation, there was found great feebleness in the respiratory murmur throughout the left lung ; but otherwise nothing morbid was detected in either the heart or lungs. He continued to improve in health up to the 22nd Jan., when, in consequence of more blood being detected in the sputa by Dr. W. S. Kirkes, who was then acting as one of my clinical clerks, auscultation was again employed, and the following note taken :—

Jan. 22nd.—He has slept well ; tongue clean ; bowels confined ; pulse 92, rather jerking, but soft ; complains of pain in the left shoulder and praecordium ; cough remains ; the sputa are white, rather flocculent, and contain a few globules tinged with blood.

Auscultation.—A harsh double friction sound is heard all over the cardiae region, also to the top of the sternum, and to some distance to the right of the sternum. It is most intense, and distinctly “to and fro,” at the base of the heart. There is increased dulness in the cardiac region, and to a slight extent beyond it. An exaggerated respiratory murmur is audible in the upper part of the right lung, with a very feeble respiratory murmur throughout the left lung.

He was ordered—Ol. Ricini 3ss. statim. Hirud. x. re-

gioni cordis. Hydrarg. Chlor. gr. ij. cum Pulv. Ipecac. Comp. gr. v. 6tâ quâque horâ. Rice milk diet.

Leeches were applied to the chest on each of the two following days; the mercurial pills were continued: no change took place in the general symptoms or in the auscultatory signs of pericarditis.

Jan. 25th.—There is mercurial foëtor of the breath, and the gums are slightly tender. He has passed a good night; the cough is troublesome; the sputa more abundant; pulse 108, soft and feeble. The friction sound remains, having become more intense and more decidedly a "to and fro" sound towards the apex of the heart. The dulness on percussion continues the same. No change in the auscultatory signs of respiration. He was ordered a blister to the chest; to take the calomel and opium every night, and an expectorant draught three times daily.

On the 27th, the friction sound was less harsh, and heard less extensively towards the base of the heart and upper part of the sternum; it remained unaltered about the apex.

On the 29th, the pericardial friction sound was less harsh and of more limited extent; the friction sound with the diastole becoming less audible, and having a softer, moister character.

On the 31st, the intensity of the friction sound had still further subsided, and it could only be heard in the proper cardiac region; there was great dulness on percussion in the same space, but fair resonance immediately around it.

The mercurial pill at night and the expectorant mixture were still continued.

Feb. 3rd.—A very faint soft friction sound may still be heard in the cardiac region, but the sounds of the heart are audible through it. Dulness on pereussion over the præcordium is diminished. There has been no prominence of the cartilages of the left ribs from the commencement; indeed, the cartilages appear depressed: this peculiarity has perhaps arisen from the constant pressure of a French organ, which he has been in the habit of carrying about with him.

Feb. 6th.—The friction sound is no longer audible, having been detected during fifteen days. Both sounds of the heart are free from murmurs. The dulness in the praecordium is more limited. He is quite free from pain; his cough is troublesome; the pulse 100, small and soft.

The mercury was discontinued.

Feb. 19th.—He complains of weakness and nocturnal perspirations; pulse 120, feeble and irregular; cough continues troublesome; his appetite is bad.

R. Quinæ Disulph. gr. j. cum Acidi Sulph. dil. $\frac{1}{2}$ v. ter die. Port wine $\frac{1}{2}$ iv. daily.

March 4th.—He sleeps well and takes his food with appetite, nevertheless he loses flesh and has a permanent flush on the checks; the pulse 120, feeble; the cough continues; the sputa consist of a clear mucus, having disseminated through it specks of pearly-looking matter, with occasional streaks of blood.

Auscultation.—Exaggerated coarse respiratory murmur on the right side. Inspiratory murmur feeble, and expiratory murmur prolonged and loud on the left side. Impaired resonance on percussion over the upper third of the left lung.

He continued the Quinine mixture, with an opiate at bed-time and the occasional employment of counter-irritation on the chest, without any material change in the symptoms of advancing phthisis until the 22nd of April. It was then remarked that his countenance was more anxious, his circulation more accelerated (pulse 116), and his nights had become restless. Upon auscultation, a friction sound was audible beneath the left mamma, extending round to the base of the same lung; there was also a diminished respiratory murmur with dulness on percussion below each scapula. On the following day he complained of pain about the right mamma, and, upon auscultation, a rubbing sound was audible between the clavicle and mamma. It was now evident that he was suffering from double pleurisy. These friction sounds accompanying the respiration varied in intensity and extent during the twelve succeeding days. At the same time the

dulness on percussion, posteriorly, increased from below upwards. This attack of pleurisy was treated by one abstraction of blood by cupping from the back, by the application of two blisters, and by small doses of calomel and diuretics.

On May 6th the pleural friction sounds had quite disappeared, having existed fourteen days, but the physical signs of extensive effusion on both sides of the chest were more decided. Blisters were again applied to the chest, and more stimulating diuretics employed.

On May 9th, the respiratory murmur was coarse and exaggerated in the upper lobes of each lung, and altogether absent in the lower lobes ; there was considerable dulness on percussion over the lower half of both lungs.

On May 10th this patient died, without any material change in the symptoms.

The body was examined twenty-eight hours after death ; it was much emaciated, and the limbs were rigid.

Lungs.—The right pleura was about two-thirds filled with bloody serum ; the left pleura about one-third filled with serum, in which were floating a few shreds of coagulated lymph. The right lung was much compressed and shrunken ; the free surfaces of the right pleura were covered with a thick layer of false membrane : this latter contained numerous opaque yellowish tubercles, about the size of millet seeds, deposited singly and in groups. The surface of the false membrane was slightly reticulated ; there were numerous adhesions, some firm and others soft, between the opposed surfaces of the pleura ; at the base of the lung the pleura was firmly adherent to the diaphragm. The substance of the lung was tough, inelastic, and contained very little air ; there were a few small groups of recent tubercles in the upper lobe, less numerous in the middle and lower lobes.

The left pleura was in the same state as that on the right side ; the upper lobe of the left lung contained plenty of air ; the lower lobe was rather compressed : a few groups of tubercles, of the same description as those on the right side, were observed throughout the lung, but most numerous towards

the apex. The absorbent glands about the trachea and larger bronchi were much enlarged, and filled with tuberculous matter.

Heart.—The surfaces of the loose and reflected pericardium were rendered entirely and firmly adherent by a layer of lymph, studded with minute tubercles. The external surface of the pericardium was also adherent to the pleura by a false membrane, in which were deposited tubercles similar to those in the false membrane lining the pleura. The cavities and valves of the heart were natural.

Abdomen.—The peritonæum was very much thickened by false membranes, in which were deposited numerous crude tubercles of larger size than those of the pleura. The convolutions of the intestines were agglutinated into a mass by adhesions. The liver was covered with a thick, firmly-adherent false membrane; its substance was healthy. The spleen contained a few tubercles, but was otherwise healthy. The left kidney congested; the right healthy. The brain was not examined.

Andral has related a case of chronic pericarditis* complicated with double pleurisy, which is very analogous to the foregoing. The symptoms of cardiac disease were very obscure; and although pericarditis was complicated with double pleurisy, and both disorders went through their various stages up to the fatal termination, there was very little complaint of pain by the patient. Nothing but the physical signs revealed the extent of thoracic disease during the life of the individual.

In Andral's case, there was found a layer of false membrane, an inch in thickness, between the free surfaces of the pericardium; and in this false membrane were discovered numerous small tubercles, some crude, and others softened. The left pleura was distended with sero-purulent effusion; while the right was agglutinated by false membranes, in which were detected numerous tubercles similar to those deposited within the sac of the pericardium. There was also

* Clin. Med., vol. i., Maladies du Cœur, obs. x.

abundant serous effusion in the peritonæum. The state of the lungs is not recorded, although they are reported as having lesions in them.

The particulars of the following case were communicated to me by my friend, Dr. Baly. The patient was only occasionally seen by myself during his last illness :—

Case 2.—John C——, ætat. 19, of a leucophlegmatic complexion and habit, but of rather a strong frame of body, had enjoyed good health previous to his confinement in Millbank Prison. He had been already more than two years in that prison, when, on the 12th of Feb. 1842, he was attacked with severe dysentery. From the acute stage of the dysenteric attack he recovered under a mercurial treatment (calomel combined with opium); but on returning to a diet of animal food, and before he had quitted the infirmary, diarrhoea, unattended with pain or any feeling of illness, came on. The alvine evacuations were almost totally devoid of bile, and for this reason moderate doses of mercury, combined with opiates, carminatives and astringents, were given to check the diarrhoea. This relapse took place in the first week of March 1842. No permanent benefit was derived from the remedies employed, although the diarrhoea was from time to time, for a day or two, arrested by the astringent medicines; but it recurred as often as the use of them was remitted, and at the same time the patient became more and more pallid, thinner, and feeble. About the 20th March he was ordered to take the Sulphate of Iron with Infusion of Calumba and Tincture of Opium every eight hours, and some brandy was allowed him. In spite of these measures he became weaker.

On the 30th March, it was remarked that, in addition to his increasing debility, the respiration was hurried and the pulse 108. The chest was examined by auscultation, and nothing but a rather exaggerated vesicular murmur was detected. Attention was not particularly directed to the state of the heart, for he had no pain in the praecordial region, did not complain of palpitation, and the frequency of the pulse

was regarded as a consequence of the hurry of respiration, and the state of general debility. During the week following, the symptoms remained the same in kind, but became exaggerated in degree. The debility and dyspnœa rapidly increased, and the pulse, concurrently with the respiration, was more accelerated. No increase of diarrhœa occurred, to account for the rapid failing of the vital powers. He died on the afternoon of the 5th of April 1842.

Upon examination of the body after death, the colon was found extensively ulcerated ; the largest ulcers were not more than a quarter of an inch in diameter, and their edges were devoid of vascularity. Both lungs were thickly studded with tubercles, for the most part in the yellow crude state, varying in size from a millet seed to a small pea, not in masses or clusters, but pretty equally disseminated throughout all parts of those organs.

The two opposed surfaces of the pericardium were firmly attached to each other, over a great extent, by a thick deposition of firm false membrane. Where the surfaces of the pericardium were not attached to each other, they were covered with thick layers of false membrane, which had a somewhat reticulated texture. The whole substance of the effused lymph was stained of a deep red colour ; coagulated blood and bloody serum were intermingled with it at various points. In one part near the base of the heart, where the false membrane was thickest and firmest, upon separating the layers of the pericardium, three or four small yellow tubercles were distinctly recognised imbedded in the lymph. Their size was about that of a grain of pearl barley.*

A drawing of the heart was made on the day after the death of the patient, and is preserved in the Museum at St. Bartholomew's Hospital.

* I much regret that, from an accident, the false membranes in other parts of the pericardium were not examined, so as to determine the amount of tubercular deposit there ; but it appears probable that a similar condition existed in all parts.

Case 3.—John P. K., *aet. 18*, a delicate man, of spare habit of body, was admitted under my care into St. Bartholomew's Hospital on May 30th, 1842, and gave the following account of himself. He was a native of Brighton, and had no regular occupation. He had been sentenced to the hulks in November 1840, and had remained there until July 30th, 1841, when he was transferred to the Millbank Penitentiary. His health had been good, according to his own statement, up to September 1841, when he began to suffer occasional attacks of diarrhœa, and on May 9th, 1842, he was placed in the Infirmary of the Prison, on account "of an increase of diarrhœa and general feelings of illness." During the following five days he was treated by Dr. Baly for the intestinal affection, when that physician observed an unusual dulness on percussion over the praecordial region, and, on auscultation, a friction sound in the same situation was distinctly audible. Calomel and opium were administered to a considerable extent, but the gums were not affected by the mercury. The extreme weakness which this patient now began to manifest, and the continuance of diarrhœa, induced Dr. Baly to suspend this treatment, and to order Ferri Sulph. gr. j. cum Pulv. Opii gr. fs. ter die; also Brandy 5ij. daily.

His strength appeared to be rapidly failing, when his pardon was granted on May 30th, and he was admitted under my care into St. Bartholomew's Hospital the same day. He was much exhausted by the removal, and presented the following symptoms on his admission:—The countenance pallid, and rather anxious; the voice weak; pulse 128, small and weak; tongue clean and moist; bowels much relaxed; respirations 28 in a minute, hurried, but not difficult. He complained of his extreme weakness, and of a short dry cough, but of no pain, except when pressure was made in the intercostal spaces in the cardiac region or beneath the margins of the ribs in the left hypochondrium. Upon auscultation, a friction sound was heard over the praecordial region and quite

across the sternum to the right side. The respiratory sounds were healthy.

He was ordered, in reference to the diarrhoea, Ferri Sulph. gr. j. c. Opii gr. fs. 6tâ quâque horâ, and a diet of bread, arrow-root, two eggs, milk, and 3vj. of port wine daily.

He slept well the night of his admission, and on the following day his condition was unaltered, excepting that his bowels had acted only once in twenty-four hours. The pericardial friction sound remained the same. He was ordered Hydrarg. Chlor. gr. v. c. Pulv. Opii gr. j. horâ somni.

June 1st.—Pain was still felt upon pressure in the cardiac region: respiration natural, pulse 112: bowels not open.

The friction sound was less intense, and the proper sounds of the heart could be heard through it. Dulness on percussion in the cardiac region scarcely more than natural. He was ordered Hydrarg. Chlor. gr. iij. c. P. Opii gr. fs. horâ somni. Olei Ricini 3fs. eras mane.

June 2nd.—The friction sound could no longer be detected, having been heard during twenty days; but the dulness on percussion was rather greater than normal in the cardiac region.

From this date he continued to progress satisfactorily, and on June 9th, when he quitted the hospital, contrary to my advice, there was no pain, dulness on percussion, nor morbid sound detectible by auscultation. His weakness was still considerable when he left the hospital.

Although the event, in this last case, did not allow us the opportunity of verifying our diagnosis of pericarditis, nor of confirming our opinion that it was an instance of tubercular pericarditis, still the history of the individual, the circumstances under which the illness arose, so perfectly analogous to those which accompanied the preceding case; the absence of any rheumatic symptoms, and the favourable progress of the patient, when he was removed from the influence of various causes which had long been depressing his spirits and his constitutional powers,—all these circumstances appear to

justify the opinion already expressed as to the nature of the disease with which this young man was affected.

The cases of tubercular pericarditis here recorded are but three; but it must be remembered that all our knowledge of disease, which is worth the title of knowledge, is derived from the observation and comparison of cases. In rare diseases, the experience of the individual physician must be small; and one of the great advantages of this Society is, that, from the assemblage of numbers and from their accumulated experience, a more complete history of a disease may be compiled than can be drawn up from the limited number of facts in the possession of any individual member.

Before concluding this communication, I will venture upon a few pathological remarks on tubercular pericarditis. First, with respect to the circumstances which appear to favour the development of tubercular pericarditis. I have no statistical records of the cases of phthisis pulmonalis, which have passed under my notice at St. Bartholomew's Hospital, and which would enable me to estimate the frequency of tubercular affections of the pericardium in phthisis. Louis reports that, in 112 fatal cases of that disorder, he found the pericardium covered with false membranes three times, but he does not state whether tubercles were looked for in the false membranes. It is probable that many more phthisical patients must have passed under his notice who were similarly affected, although the disease was never suspected nor looked for, because simple pericarditis often occurs without being detected during life, and does not commonly end fatally. So far as any conclusion may be drawn from a limited number of cases, it would seem that tubercular pericarditis is not so much to be looked for in ordinary cases of phthisis, where tubercular disease, in the form of granulation and infiltration, invades the different lobes of the lungs, and rapidly runs through its disorganizing processes; but this disease is rather to be anticipated when individuals, previously healthy, are exposed for a length of time to the influence of the most

powerful exciting causes of the tubercular diathesis. Dr. Baly, in his elaborate and valuable essay, "On the Mortality of Prisons," (Transactions of this Society, vol. xxviii.), arrives at the conclusion, "that not merely pulmonary phthisis, but tubercular disease in all its forms, is the frequent result of the long-continued influence of imprisonment on bodily health." The fact that two cases out of the three just recorded, occurred among persons who had long been confined in Millbank Prison, tends to corroborate the opinion advanced by Dr. Baly. The circumstances under which the first related case occurred, are not very dissimilar to those of the other two. The patient was an Italian musician, who, having been tempted to leave his sunny clime, found himself doomed to spend a northern winter, and such a severe one as that of 1843-4, wandering through the dreary streets of this metropolis, daily wearied with his burthen, dispirited, badly fed and clothed, and passing the long winter nights in a close, ill-ventilated lodging. Such is the class of persons among whom this disease should be sought after or suspected, and particularly if the constitutional symptoms of tubercular disease are well marked, although the physical signs of pulmonary disease are but slight and equivocal.

Other questions of pathological interest here present themselves, and are worthy of investigation. What relation do the tubercles and the inflammatory fibrinous exudation in the pericardium bear to each other? Which lesion is cause and which effect? Or do both lesions result from contemporaneous morbid actions?

Laennec and Rokitansky (*loc. cit.*), who have both described this lesion of the pericardium, have, I think, erroneously supposed that the tubercles are deposited in the false membranes of pericarditis, subsequent to, and as a consequence of, the inflammatory process. Whereas, it appears to me more consistent with our present knowledge of the exciting causes of inflammation and tubercle respectively, to regard the inflammation of the serous surface of the pericardium as resulting from the previous accidental de-

posit of tubercle on the membrane, and to ascribe the production of the tubercles to the operation of causes quite independent of inflammation, and acting prior to the inflammation. Rokitansky, indeed, broadly affirms that tubercles occur in the pericardium most commonly as a product of inflammation, and very seldom under any other form. His explanation of the formation of tubercles on the pericardium is nearly as follows:—"An attack of pericarditis deposits an exudation, the peripheral coagulated portions of which undergo either complete or only partial conversion into tubercle. When a second attack of pericarditis ensues, it frequently happens that the deepest or oldest layers of the exudation become converted into tubercle; or, it may be that the most recently-deposited layers of fibrin, having become tubercular, are covered by a villous shreddy substance deposited from the fluid effusion. The tubercles, which are often very large and aggregated into masses, are sometimes deposited quite close upon the muscular substance of the heart. These tubercles seldom go on to softening, for most commonly death soon occurs, either from the pericarditis itself, or from deposit of tubercles elsewhere, or from the general cachexia."

Laennec has also advanced a similar opinion, and gone so far as to state that these tubercles, deposited in the false membranes after an attack of acute pericarditis, cause the inflammation to become chronic, thus regarding them as an effect of acute and a cause of chronic inflammation.

It appears to me a more simple and philosophical explanation of the phenomena, and one more in accordance with our present knowledge, to state that the tubercles are deposited in the first instance as a result of tubercular cachexia, and that these foreign bodies then excite an inflammation of the serous membrane on which they are deposited, and that fresh crops of tubercles springing from the same state of constitution keep up the inflammatory action, and it is thus that the acute is converted into chronic pericarditis. The situation of the tubercles, as described by Rokitansky, in close contact with, and almost imbedded in, the muscular substance of the

heart, points out that they are deposited there prior to the fibrinous exudation with which they are covered. A similar explanation of the pathology of that variety of chronic peritonitis which is connected with the presence of miliary or tubercular granulations on the peritonæum is adopted by Dr. Watson, who states (Lect. 66, 2nd edition) that he is in the habit of regarding the tubercular granulations as the cause and not the consequence of the peritoneal inflammation with which they are found associated. Most pathologists admit that tubercles, like other foreign bodies, may act as irritants to the tissues with which they are long in contact, and thus excite inflammation. Andral,* Lombard of Geneva, Carswell, and some others, explain the softening of tuberculous matter through the intervention of a purulent secretion poured forth from surrounding inflammation excited by the tubercular deposit. It is not surprising, then, that inflammation should be excited on the surface of a serous membrane by the deposit of tubercle there, and that such inflammation should lead to an abundant fibrinous exudation.

If such be the correct explanation of the origin of this variety of pericarditis, I think the ordinary treatment of that affection, viz., by repeated abstraction of blood, and by bringing the system under the full influence of mercury, is quite inappropriate. Such remedies, by impairing the powers of the constitution still further, would certainly favour the continued development of tubercle in the pericardium and elsewhere, inasmuch as it is from cachexia that a similar condition of other serous membranes is observed in the same individual. Tubercular pleuritis or peritonitis is frequently found co-existing with the tubercular pericarditis, and it is quite uncertain which serous membrane may have been the primary seat of deposit of the tubercles. If depletion be practised in these cases, a very moderate abstraction of blood will be sufficient; mercury should be used sparingly, and every precaution taken to prevent ptyalism. Counter-irritation by

* *Precis de Pathologie*, vol. i. p. 415.

blisters over the cardiac region, saline diuretics with compounds of iodine, and the speedy removal of the patient from the influence of those causes which are known to favour the development of the tubercular cachexia, will probably be found the most efficacious remedies in obviating the fatal consequences of the chronic pericarditis produced by the irritation of tubercular deposit.

SUCCESSFUL
REMOVAL OF AN OVARIAN TUMOUR,
COMPLICATED WITH PREGNANCY.

BY H. E. BURD,
SENIOR-SURGEON TO THE SALOP INFIRMARY.

COMMUNICATED BY JAMES PAGET.

Received Feb. 1st—Read March 23rd, 1847.

THE Profession being still divided in opinion as to the propriety of removing ovarian tumours with the knife, I take the opportunity of communicating to the Royal Medical and Chirurgical Society the history and result of a case operated upon during pregnancy; an occurrence which, as far as I know, has not before been recorded, and therefore possesses special interest.

Ann Jones, *ætat.* 25, rather above the middle size, and inclined to be stout, was admitted into the Salop Infirmary on the 28th of February 1846. She has been married four years, and had three children; and has always enjoyed good health until her last confinement (seven months ago), after which she first detected enlargement of her abdomen. She thinks she is nearly as large now as before her delivery. Her last child is strong and healthy; the labour was not a difficult nor protracted one, and her previous confinements were all favourable. She says that in about a month after her last confinement she perceived a hard tumour in the abdomen, which has remained much the same in size ever since; she also says that it moves about when she changes her position.

March 3rd.—The measurement of the abdomen over the

umbilicus is 37 inches; its shape is irregular, and on the right side a considerable tumour may be distinctly felt. Its attachments appear to be very loose, for it may be moved about with great ease. The fluctuation through the abdomen generally is very imperfect; and in the tumour itself, upon isolating and carefully manipulating it, no fluctuation is perceptible.

April 1st.—She is much the same as regards the ovarian disease, and her general health seems not at all affected. Tongue clean; pulse 80, but small; bowels regular; urine natural and without sediment.

9th.—No perceptible change in the size or hardness of the tumour.

Up to this date she was under the care of Dr. Johnson, physician to the infirmary, who now requested me to see her, which I did on the 10th; and having learned that she had experienced but little relief from any of the remedies taken, all means likely to ameliorate her condition having been adopted, I recommended that she should omit all medicines, and only wear an elastic bandage to support the tumour. She acted upon this advice, and went home, being told at the same time that if the tumour increased she had better return to the infirmary.

On the 5th of September, after five months' absence, she returned to the infirmary, much increased in bulk. The measurement over the umbilicus was then 45 inches, and from the ensiform cartilage to the pubes 22 inches; there was very distinct fluctuation through the abdominal parietes. The appearance above the umbilicus was irregular, and, on manipulation, a sensation of solidity was transmitted to the touch. Her dyspnoea was very urgent, and she was unable to lie down. She said that she had menstruated regularly since she returned home, and that the last time was about six weeks ago, when the discharge was profuse. The breasts were flaccid, and the areolæ were not darker than is usually remarked in women who have had children. She felt confident that she was not pregnant, having none of the symptoms

she used to have on former similar occasions. Examination, per vaginam, afforded no proof of her state; in fact, the os uteri could not be reached by the finger. The usual stethoscopic signs of pregnancy were also sought for, but not detected in consequence, no doubt, of the situation of the tumour. Thus the woman's own history, and all the direct local signs which could be obtained, afforded no grounds whatever for suspecting that she was pregnant.

The opinion of the medical staff having been requested, it was unanimously agreed that the case was of a nature which justified an attempt to remove the tumour by operation.

The patient was made fully aware of the possible consequences of this proceeding; and the temporary relief she would experience by tapping was likewise explained to her, as also the uncertainty and danger of that operation, which has of itself in many cases proved fatal, and rarely gives more than partial relief, and that only for a short period. Under these circumstances, she came to the determination to have the tumour removed at all risks, and begged that an early time might be fixed for the performance of the operation.

On the 15th of September, at noon, the bowels having been freely evacuated on the previous day by a dose of castor oil, I proceeded to operate. The temperature of the room having been raised to 72° (Fahrenheit), the patient was placed on a convenient couch, her back being supported by pillows whilst her feet rested firmly on a footstool. The pulse before the operation was 98. An incision, sufficiently large to admit the finger, was first made through the parietes of the abdomen, about half way between the umbilicus and pubes, by which a small quantity of clear fluid escaped. The surface of the tumour was now exposed. By the introduction of the finger as far as possible, and by passing it round the tumour, no adhesions could be felt. The incision was then extended upwards to within about an inch and a half of the ensiform cartilage, and downwards to the pubes, and still no adhesions presented themselves. I now passed a large trocar into the

largest cyst, and drew off three gallons and a quart of thick, glairy, highly albuminous fluid, which reduced the tumour to a convenient size for passing through the aperture; previous to this it was too large to allow of its being drawn out, although the incision was as extensive as possible.

On drawing the tumour forward so as to examine its pedicle, the uterus was brought into view in a gravid state, and was supposed to have reached the third or fourth month of pregnancy. The pedicle of the tumour was extremely thick and broad, requiring to be tied in three different compartments with strong twine ligatures. The bulk of the pedicle made it so difficult, even with this division, to draw the ligatures sufficiently tight, that when the tumour was separated from the pedicle the upper and outer one slipped, and had it not been for the prompt assistance of my colleagues, a fearful haemorrhage would have ensued. Another strong ligature was, however, immediately passed through the pedicle, and being tied, prevented further bleeding. It was yet thought more safe to search for the mouths of the vessels, and place a silk ligature round each separately. This being done, and the parts having been thoroughly cleaned, the margins of the incision were brought into apposition by a few interrupted sutures; a cordial was then administered, and the patient was placed in bed. The lips of the wound were now accurately brought into contact by other sutures; and adhesive plaster, a few compresses of lint and a bandage completed the dressing. The pulse was at this time 88, small and weak.

Ordered—T. Opii 3*j.* statim.

On examination of the tumour, after removal, it was found to consist of a multiplicity of sacs surrounded in some instances by solid matter: all of these sacs were situated anteriorly, which circumstance, no doubt, gave the swelling its irregular form in front. The fact of the tumour being sacculated would have rendered it impossible to empty it by tapping, and of course the solid matter would have remained after that operation. The walls of the cysts were

thick and firm, and the whole tumour, with its contents, was computed to weigh not less than 50 lbs.

3 P.M.—Complains of pain in the back; countenance expressive of pain. Pulse 90. Has been sick once, but vomited only a small quantity.

6 P.M.—Has had half an hour's sleep, and has taken a little gruel. About 4 oz. of urine drawn off by catheter.

7 P.M.—Since last note, has had uterine pains, occurring every five or ten minutes. Ordered—T. Opii ʒj. ex mist. camph. stat.

16th—5 A.M.—Still complains of pain, especially in the left iliac fossa. Sleeps only at very short intervals. Catheter introduced. Ordered—T. Opii ʒſs. ex aquâ statim.

9 A.M.—Countenance more anxious. Still complains of pains in the back and side. Has had about two hours and a half sleep during the night; speaks cheerfully; pulse 108, rather contracted; tongue moist, with a brownish fur on dorsum. Has had no return of the vomiting.

4 P.M.—Breathing a little hurried. Pulse 130, small and contracted. Skin hot. A little mucus passes per vaginam. The uterine pains having returned, the os uteri is beginning to dilate. Pain still continues in the left iliac region, with considerable tenderness. Ordered—Venæ sectio ad ʒiv.

10 P.M.—The blood drawn at 4 P.M. is now buffed and cupped. Pain relieved. Countenance improved. Pulse 120, soft and compressible. Has taken during the day a little tea and arrow-root.

17th—7 A.M.—Has had a continuation of the uterine pains during the night, which have increased lately.

Soon after this, abortion took place: the labour was easy and not protracted. The foetus presented naturally, and was alive when born. About an hour afterwards the secundines came away, x. grs. of ergot of rye having been administered, as the uterus seemed inclined to be sluggish. No haemorrhage ensued, and the woman bore her labour better than could have been expected.

1 P.M.—The abdomen is rather tympanitic and tender, breathing oppressed, and she has been slightly sick. Abdominal bandage was slackened. Ordered—Liq. Opii Sed. η xx. stat.

5 P.M.—Countenance anxious, and a troublesome cough has come on. Has passed water through the day without assistance; there is no appearance of any lochial discharge.

8 P.M.—There is now much tympanitis, with frequent hiccough, and pain in epigastrium, accompanied by extreme restlessness. To take small quantities of brandy-and-water, with ammonia; also Pulv. Opii gr. iij. statim.

18th—10 A.M.—Has had a good deal of sleep, and feels relieved. Pain in epigastrium quite gone. Countenance improved. Pulse 116. Dressed the wound, which looks well and is to a considerable extent united. Removed several of the sutures, and changed her linen, which seemed to refresh her. Takes tea, coffee, beef-tea and new milk.

2 P.M.—Suddenly taken with faintness and loss of vision. Pulse fluttering, countenance very anxious. Ordered—Ammon. Sesquic. gr. v. Liq. Opii Sed. η xx. statim. Ext. Opii gr. iij. post $\frac{1}{2}$ horam.

8 P.M.—Much more comfortable; a coagulum of about 2 oz. has passed from the uterus, with some lochial discharge.

12 P.M.—All pain has ceased. Complains of great exhaustion. Feels disposed to sleep, but wakes up in a fright. Tongue moist, urine abundant. Bowels not opened. Ordered—Fellis Bovis, gr. x. Pulv. Opii gr. iij. statim.

19th—4 P.M.—Had some comfortable sleep during the night; but is now restless and uneasy. Cough very troublesome. Discharge through the wound has become very much more serous and more profuse; pulse rapid. At 6 P.M. she was ordered—Fel. Bovis c. Pulv. Opii gr. iij.; and at 9 P.M. the following draught: T. Opii η xxx. Spt. Ammon. Ar. η xxx. Inf. Aurant. co. $\frac{3}{ij}$. stat. sum., which was given with the ox-gall, and repeated at midnight.

20th—2 A.M.—Restless still, and sighing. Complains

of great pain through the hips. Perspiring about the face, with cold extremities. Throws off the bed-clothes. To take small doses of brandy. Ordered—Pulv. Opii gr. iiij.

7 P.M.—She slept a little in the early morning, and the bowels were relieved by an injection of castor oil, turpentine and gruel; but she has had a very uncomfortable day: features much sharpened, extremities getting cold; and spasmodic contractions of the diaphragm have lately come on. Pulse 120. Ordered—Fellis Bovis gr. x. Pulv. Opii gr. iiij. stat.

10 P.M.—Is asleep under the influence of the opiate. Has constant jactitation, and is much exhausted. Extremities cold, breathing heavy and slow. Countenance pinched and covered with perspiration. Pulse 130, small. Ordered—Repetatur Fel. Bovis c. Opio.

It is scarcely necessary to continue the frequent reports by which it has been thought desirable to mark the progress of the case up to the last date. Suffice it to say, that some days elapsed before I considered my patient out of danger. Thus, on the 21st, there was tenderness and distention of the abdomen, accompanied by much restlessness; and these symptoms continued, with some intermission, through the following day. The ox-gall and opium, together with fomentations to the belly and injections of beef-tea, were then found serviceable. On the 23rd, the pulse continued rapid and the bowels were relaxed. Her diet was improved, and a tonic mixture was prescribed, containing also some aromatic confection; and an opiate injection was administered. On the 25th, my patient was much improved in every respect; and had no further recurrence of the symptoms which had previously so much distressed her. On October 8th, three of the ligatures came away, and the remainder separated spontaneously towards the end of the month. On November 6th, the wound was quite healed, and Mrs. Jones was able to walk about. She left the hospital on the 15th of this month, and was soon afterwards able to engage, without inconvenience, in her usual domestic duties.

I shall confine my concluding remarks to the peculiarities presented by the case I have just recorded. I did not consider the operation justifiable on the patient's first admission into the infirmary, her general health being then undisturbed, and enabling her to attend to her domestic duties without inconvenience. With regard to relief by tapping, my impression from the first was, that the tumour consisted of several sacs, and that some of them invested solid matter which, of course, could not be removed by the trocar. The fact of her being pregnant is of course a circumstance of no small interest; and this interest is greatly increased when it is remembered, that all the means which had been employed to ascertain the condition of the uterus went to prove the absence of pregnancy. On examination of the aborted placenta, it was evident that there had been a partial separation at one point, with a considerable clot of blood adhering, and that haemorrhage had ensued which was mistaken for menstruation.

Here, then, is an occurrence calculated to mislead the most vigilant, showing not only the uncertainty of placing any dependence on a patient's own description, but also the fallacy of resting upon a patient's history of menstruation, as an unerring test by which the non-existence of pregnancy may be determined.

In the after-symptoms, one cannot but be struck with the absence of peritoneal inflammation, four ounces of blood being all that it was thought necessary to take away. The most important and alarming symptom was the frequent occurrence of collapse. Opium, ammonia and brandy immediately relieved this condition, and was had recourse to largely, with the very best result. It was, in fact, especially interesting to mark, during the whole after-treatment of the case, how opium immediately relieved all the symptoms of collapse. The patient was no sooner from under its influence than she became restless and uneasy; but, on a repetition of the opiate, she uniformly rallied, and remained comfortable until another dose was called for by a recurrence of the symptoms.

At one time, I entertained a fear that the constant and profuse watery discharge from the wound would so weaken my patient, as to render her constitution unable to hold out during the period of convalescence. This discharge, however, gradually ceased; the wound assumed a healthy appearance and rapidly healed, after which she quickly improved, daily gaining strength and flesh.

To decide on the propriety of performing the operation of ovariotomy must always require the exercise of careful judgment and sound discrimination; for if, in most of the larger operations of surgery, many unforeseen complications may present themselves, in this operation more especially we must be prepared for them.

Whether the record of the present case will tend to strengthen or weaken the confidence of surgeons in undertaking the removal of diseased ovaria by the knife, I cannot take upon myself to say; but I think it is useful and satisfactory to know, that a complication such as I have detailed does not necessarily involve a fatal result.

CASE
OF
ENLARGEMENT OF THE LEFT MAMMA.

BY W. E. IMAGE, F.R.C.S.,
SURGEON TO THE SUFFOLK GENERAL HOSPITAL.

TO WHICH IS ADDED AN
ANATOMICAL AND PATHOLOGICAL DESCRIPTION
OF THE TUMOUR.

BY T. G. HAKE, M.D.,
PHYSICIAN TO THE SUFFOLK GENERAL HOSPITAL ;
AND
W. E. IMAGE.

COMMUNICATED BY ROBERT LISTON, F.R.S.,
VICE-PRESIDENT OF THE SOCIETY.

Received February 18th—Read April 13th, 1847.

SARAH HARVEY, æt. 21 next birthday, a native of Brandon, was admitted into the Suffolk General Hospital, April 15th, 1845, with an enormous enlargement of the left breast.

Previous history.—Very little indeed was to be gleaned, either as to the nature of the disease or the stages of its formation, from the patient's history prior to her admittance into the hospital. Her health appeared to have been generally good: she did not remember to have been ever ill until three years ago, when she quickly recovered, and again resumed her usual avocation of household servant. She was quite sure her illness did not then influence the breast, although not aware what was its character. About two years since,

for the first time, she observed a red mark, about the size of a shilling, just above the nipple, and that the breast was enlarged: it was painless, even under pressure. She had not observed the enlargement of the breast until the red patch attracted her notice: the catamenia were natural. She continued, for two months after this time, free from any pain in the breast. As soon as it became painful, it appeared that leeches and cold lotion were employed. The breast continued gradually to enlarge, the pain remaining the same; which, however, was not severe. Iodine was also employed fully, but ineffectually.

She remembered having struck her breast some time since with a pump handle, but did not suffer from the blow at the time; and she did not connect the accident in any way with her present disease. In fact, she was unable to remember whether the breast had begun at that time to enlarge.

Actual state, April 15th, 1845.—Breast pendulous: there is a blue, nævus-like spot just above the nipple, about the size of a half-crown piece, and several smaller ones of the same description in its vicinity. A general bluish or slate-colour characterizes the entire surface of the breast. The skin itself, except in the places above-mentioned, is normal, and its leaden hue disappears during pressure. The diseased mass measures around its base fifteen inches; vertically across, nine inches; horizontally across, eleven inches. By gradual pressure, it admits of being reduced to at least one half its bulk. The veins appear enlarged. There is no pulsation, no murmur. When the tumour is reduced by pressure, the patient complains of fulness and heaviness in her head; and on the pressure being withdrawn, pallor and faintness supervene: these manipulations are productive of scarcely any pain. The patient has the fresh and healthy appearance of a country girl; the catamenia are regular and natural, and no difference is experienced in the breast at the menstrual epoch: indeed, with the exception of occasional faintness of an alarming kind and deep mental depression, the constitution is healthy.

The treatment employed was pressure by an air-cushion

within a metallic hemisphere, so as to bear equally upon every part of the breast. The plan was continued three months, but no advantage resulted from its adoption. During its use, the pressure afforded a feeling of support to the part; on its withdrawal, the mamma quickly became distended again, and great faintness invariably followed the return of blood to the tumour. Meantime, in spite of the diminution of bulk by pressure, which was effected by the treatment, the disease itself actually advanced, and in September, five months after admission, presented the following appearances. The patches of discolouration, previously described, had enlarged and become blended, new detached spots had appeared, and the nævus-like discolouration had attained to at least six or seven times its former size, as represented in the Plate. This morbid superficies presented an irregular form, consisting of conjoined patches and isolated spots, having the character of nævus. The parts originally red had become purple, and those newly developed were red. The nipple had become almost obliterated, and the areola was obscured by the invasion of the morbid process. The spot primarily affected had become the seat of venous dilatation, so conspicuous as to form a prominent feature, and its outer tegument was so attenuated as to excite fear of its speedy rupture.

On September 14th, 1845, I conducted my patient to London, and accompanied her to the houses of several surgeons, many of whom I was so fortunate as to find in town. No material difference of opinion existed as to the pathology of the tumour, but much as to the best mode of meeting the exigencies of the case by surgical art. Some high authorities suggested amputation of the entire breast, opposing whatever amount of haemorrhage might occur, by having assistants ready with sponges, tenacula and gallic acid, together with such other expedients as might counteract the retraction of branches of the internal mammary artery, and entrance of air into the veins. Again, it was suggested to introduce single threads through the breast, with a hope of ultimately obliterating the cellulated structure of the tumour. And, finally, it was pro-

posed to take the following course, which, from a preponderance of opinion in its favour, was ultimately adopted.

To place the patient in a recumbent position, to make a vertical incision on either side of the enlarged mass, and, as far as the healthy skin would permit, to dissect back two flaps, securing the bleeding vessels as the operation proceeded ; to pass two very long and strong needles, firmly fixed in handles, through the base, so as to meet each other at right angles in the centre of the base of the tumour ; and having armed them with double ligature, to return them. The needles being detached, the ends of the ligature, eight in number, were to be firmly tied, and the tumour was thus to be strangulated.

I returned home to Bury, and my patient was greatly fatigued by the journey. She fainted at a house on the road, and continually appeared exhausted by faintness. The tumour at this time projected 7 inches from the chest, and measured 23 inches around its base, 13 inches vertically across, and 15 inches horizontally across. It was evident that it was daily increasing ; and the menstrual period having just passed away, I determined to operate speedily. There was at this time a very distinct thrill communicated from the heart.

The operation was performed on the 25th of September 1845, and lasted twenty minutes ; during the latter part of which she was faint, having lost about 14 oz. of blood, and it was evident the shock was severe during the time the ligatures were tightened. She vomited soon after she had reached her bed, and the pulse was scarcely perceptible ; after a short time, however, she rallied a little. Stimulants, mixed with gruel, as well as opiates in a liquid form, were administered from time to time, but were not retained as the vomiting returned. There was now considerable oozing of venous blood, particularly from the inferior portion of the wound through which one of the ligatures had passed. It was arrested by gallic acid and pressure. Opium and beef-tea were administered by the rectum.

Sept. 26th.—Passed a sleepless night. The vomiting still continued, as well as some degree of venous oozing. The

pulse was rapid and small, with other symptoms of collapse, the result of shock and loss of blood. The total loss of blood amounted to about 30 or 36 oz. She sank at half-past 10 A.M., twenty-two hours after the operation.

Autopsy.—8 A.M., Sept. 27th—22 hours after death, and 44 hours after the operation.—Body was yet warm. The superficial veins of the neck appeared large and dark. The tumour black, skin detached in vesicles, and rapidly decomposing. The left pleura contained 3ij., the pericardium 3jfs., of bloody serum. The skin over the sternum peeled off with the slightest touch, and decomposition of the surrounding parts appeared to have rapidly advanced. There were no other abnormal appearances. The tumour was carefully taken off, with a portion of the ribs, for minute examination.

Anatomical and Pathological Description of the Tumour.

The external characteristics of the diseased breast have been described above; it remains to give a statement of what was discovered on dissection of the tumour.

A horizontal section of the mamma through the nipple down to the base having been made, an appearance presented itself which was calculated to mask rather than reveal the nature of the disease. It is to be recollected that the ligatures used in the operation, when drawn tight, had the effect of compressing the blood within the vessels of the mamma, and of causing it to stagnate there. The tumour thus strangled had been cut off from the circulation for a period of twenty-two hours, and had remained for a like period after death before it was dissected. The appearances, therefore, on a section being made, were those of strongly-marked congestion, caused by the operation. In the midst, however, of this artificial condition of the tumour, there existed traces of structure both healthy and morbid.

The skin was unaltered in structure, except in the places already described, where the disease was visible externally.

The adipose tissue and fibrous laminae, situated between the skin and glandular substance, were compressed together,

on the anterior aspect of the breast, into a dense tough membrane, in which there was an almost total absence of the fatty matter. At and around its base, however, the fibrous tissue was natural, except where perforated by dilated veins; and was intermixed with adipose tissue in the usual manner underneath the skin which surrounded the base of the organ.

The lactiferous ducts were observed passing from the glandular structure through the parts in front towards the nipple, but were lost in the condensed tissue before reaching it.

Vestiges of gland, varying in size from that of a millet seed to an almond, and of different forms, were scattered over the exposed surface; and, in common with other structures, were tinctured more or less by the blood which had been forced into the vessels by the ligatures.

Besides the venous apertures already alluded to, there existed in the fibrous tissue at the base of the organ, where the dilated veins penetrated, certain cells of considerable size, which were the result of the operation, as will be shown presently. Within these false cells blood was found, in a semi-fluid state and resembling currant jelly; and connected to their walls were seen the torn remains of lactiferous ducts and glandular substance.

It was ascertained by dissection that the arteries and nerves of the mamma were unaltered.

The absorbents, owing to the state of the preparation, were not examined, but there was no evidence of their enlargement. The capillary vessels, for the same reason, could not be injected.

The internal mammary vein, towards its junction with the subclavian, presented an irregular and sacculated appearance. Immediately in advance of each sacculated portion, in the direction of the heart, the vein was narrowed.

The interior of the sacculated portion of the vein was found to present a valve-like formation; the narrowed parts were thickened, and the sacculated parts were formed of one hollow within another.

The superficial veins were dilated uniformly into large sinuses.

The mammary veins, internal and external, were traceable backwards into a cellulated structure, to be described presently.

All the preceding facts were visible to the eye without assistance ; what follows was discovered by the aid of the microscope.

In whatever part of the mammary organ a section was made, and examined under water through the microscope, with a low power, the apparently uniform and glistening surface was resolved into cells. These cells were of various sizes ; and within the greater, lesser ones were visible. Into these cells all the veins of the organ were traceable ; indeed no vein was found to have any other origin, so that the cellulated structure of the tumour was essentially venous, consisting in fact of the veins situated between the capillaries and vein-trunks in a state of distention. The rupture of these cells, from pressure of blood under the ligatures during the operation, produced those large false cells already described as containing blood, pendulous fragments of lactiferous ducts, and their attached glandular substance.

The dilatation of the more minute venous structure of the organ into cells was a medium through which every tissue may be said to have become preternaturally, though uniformly, separated and distended. The effect of this was very evident in the fibrous as well as the glandular structures, which are intimately associated in the breast. By the increase of bulk to which this cell formation gave rise in the organ, the lobes of the gland were separated further apart, and these, in their turn, were subdivided into isolated lobules.

The distending force had likewise produced its characteristic effect on the fibrous structure of the organ, the tissue in question being known to involve the gland and its parts. In health the fibres of this tissue are parallel and closely united ; but when examined in the present instance, they were found to have given way to the dilated veins. Their texture was com-

pletely altered, being converted in some places into a kind of network through which the vein-cells passed. The process of formation of this cellulo-fibrous tissue may be thus described. In the first instance, the fibres are so slightly separated as to be only no longer parallel ; they are then perforated by venous cells at intervals ; and finally are separated so completely as to appear cellular.

Such were the effects, doubtless, of the formation of venous cells, itself caused by the force of the blood acting gradually and during a considerable period of time upon the venous system, the free passage through which was choked up near the junction of the left internal mammary vein with the sub-clavian. The slight disproportion between the arterial and venous circulation, resulting from the narrowed condition of the internal mammary vein, was the evident cause of the fatal disease which ensued. There was more blood supplied by the arterial, than could be carried off by the venous system of the mamma ; hence its accumulation, and the adaptation of structure to this new condition of the circulation of the organ.

The disease above described, caused probably by the effects of a blow on the trunk-vein of the left mamma, is unique, and may not occur again for a considerable time.

EXPLANATION OF PLATE II.

This plate exhibits the appearance presented by the breast of Mr. Image's patient, just prior to the operation for its removal.



C A S E
O F
C Y A N O S I S ;
W I T H
A D E S C R I P T I O N O F T H E A P P E A R A N C E S P R E S E N T E D
O N D I S S E C T I O N ;
I L L U S T R A T E D B Y T H E P R E P A R A T I O N , A N D A D R A W I N G ,
O F T H E H E A R T .

BY F. LE GROS CLARK, (SEC.)
ASSISTANT-SURGEON TO ST. THOMAS'S HOSPITAL.

Received March 12th—Read May 11th, 1847.

THE subject of this case came under my notice, a few weeks back, in the dissecting room of St. Thomas's Hospital. I have since gleaned the particulars which have enabled me to draw up the following history; and for them I am indebted to Drs. Bevan, G. O. Rees, Cohen, and Leeson, under whose care the boy was, successively, until the time of his death.

William Baker, *aet. 19*, shoemaker, was admitted into Clinical Ward, St. Thomas's Hospital, July 28th, 1846. He stated that he had been ill, with the attack under which he then laboured, two months; but that he considered his illness to have been occasioned by a strain which occurred in August 1845, a week after which he experienced pain in the region of the heart, and suffered from extreme dyspnœa. For this he became a patient at the Islington Dispensary, and afterwards went into Guy's Hospital, where he remained eight or nine months. Two months before his admission into St. Thomas's Hospital, he had haemoptysis on two or three occasions. He also stated that he had rheumatism four years ago; but, on being questioned respecting this attack, the symptoms

resolved themselves into severe dyspnoea with pain in the chest, but unaccompanied by swelling of the joints, shifting pains, or any peculiarity in the appearance of the urine.

Dr. Bevan, under whose care the boy was prior to his admission into Guy's Hospital, perfectly remembers the case, and confirms the accuracy of the diagnostic signs which I shall presently enumerate. This gentleman also believes the lad's mother distinctly affirmed, that he had always suffered from lividity of countenance and other evidences of disturbed circulation; but the boy denied that such was the case, when questioned by Dr. Cohen at a later period, although he admitted that his constitution had been ailing from early childhood; pain in the cardiac region and palpitations having prevented him from being able at any time to take much exercise, or to do any hard work. Unfortunately the friendless condition of the boy has rendered abortive all attempts I have made to prosecute this inquiry.

When in Guy's Hospital, Dr. Rees informs me that Baker "suffered occasional attacks of dyspnoea, and on exertion became livid. His extremities and nose were often cold, the former nearly always so when his attacks of dyspnoea occurred. There was a loud bruit heard below the left nipple, and also upwards from that spot and to the right, reaching over the sternum. This bruit accompanied the systole of the ventricle, and was of a loud *swishing* character." He continued in Guy's Hospital nine months, and then quitted it, without having experienced any marked benefit.

On admission into St. Thomas's Hospital, five weeks after his dismissal from Guy's, the following entries were made in their books by Drs. Liceson and Cohen, who have kindly supplied me with their notes.

"Habits apparently regular. Disposition placid. Complexion remarkably livid. Skin cold and dry. Bowels habitually torpid. Urine natural. Lips swollen and very livid: and the alæ nasi distended. Urgent cough, with extreme dyspnoea, render the recumbent posture very distressing. The superficial veins of the chest are extremely dilated; and

at times there is a jugular pulsation. Pulse quick and labouring. Does not complain of any pain.

"On percussion, the chest yields over its whole extent a dull sound, excepting over the anterior mediastinum, where the sound is clear; so that it is impossible to ascertain the size of the heart. With the stethoscope a very feeble respiratory murmur is detected, but unaccompanied by any morbid sounds.

"The impulse of the heart is natural. Its sounds are superseded by a very loud prolonged systolic bruit, heard over the whole region of the heart, but most especially over the aortic valves. The sound, however, does not extend beyond its arch in the direction of the aorta, but spreads out laterally from the point where it is heard in its greatest intensity, with an equal degree of force, along the course of the left branch of the pulmonary artery, and may be heard on that side to the very periphery of the chest. The sound on the right side is also heard in the course of the right branch; but here it has, although distinct, a more distant character, and does not reach to the same extent. There is also a systolic bruit over the apex of the heart."

The preceding account was principally furnished me by Dr. Cohen, who first saw the patient. When examined by Dr. Leeson, nearly a month subsequently, some of the symptoms seem to have undergone a modification, for he then complained of pain of a lancinating character in the chest, reaching through to beneath the scapulae; the respiratory murmur had become sonorous and wheezing in the larger bronchi, and the pulse feeble, slow, and irregular.

A lowering plan of treatment was first adopted, but was subsequently changed to one of a "gently stimulant and tonic" nature, from which he seemed to derive most relief; and his suffering was always mitigated by fine and dry weather. Bleeding appeared to afford very little relief.

During the last two days of his life, the lividity of his face, the dyspnoea and suffering, were greatly increased, and he was quite unable to lie down; he died on the 3rd of January 1847.

Autopsy.—The body generally was wasted, and the feet and lower part of the legs were oedematous. The right lower extremity was shortened and inverted ; and, on dissection, the head of the corresponding femur was found partly absorbed and resting on the dorsum ilii, the capsular ligament being much thickened. The left hip-joint was also partially affected in a similar way.

Of the abdominal viscera, the liver was large and dense : the spleen was thrice its ordinary bulk, but apparently unaltered in texture. The kidneys and membranous chylopoietic viscera presented nothing abnormal.

The adjacent surfaces of the pleura on either side of the chest were adherent throughout the greater part of their extent. The lungs were a good deal loaded with blood, and in many parts dense and mottled. There were a few small circumscribed cavities in one lung, containing softened tubercular matter.

On laying open the pericardium, it was found to contain a small quantity of limpid serum ; but it was not adherent at any part to the surface of the heart, nor did it present any other evidence of morbid change.

The bulk of the heart was decidedly great in proportion to the development of the frame generally ; but it was at once apparent that this increase in size was especially due to the abnormal capacity of the right auricle. The auricle and ventricle of either side were laid open by a perpendicular incision, extending from the base to near the apex of the heart. Some wax injection, which had been thrown upwards from the abdominal aorta, was found in the cavities ; yet, with the exception of a little thickening in the general texture of the tricuspid valve, both the auriculo-ventricular and arterial valves seemed to be healthy. The left auricle and ventricle presented nothing to attract attention, except, perhaps, a trifling attenuation of the walls of the latter.

The right side of the heart presented the following appearances :—The auricle, more particularly its appendix, was largely dilated, and the intervals between the fleshy fibres were thin and transparent. There was an oblique communi-

cation between the two auricles at the fossa ovalis ; but this I have found so commonly the case that it can scarcely be regarded as abnormal. The auriculo-ventricular communication was free and unobstructed, and the termination of the *venae cavæ* presented nothing unusual. On comparing the walls of the two ventricles, it was apparent that the usual relation of development was reversed, the parietes of the right cavity being nearly twice as thick as those of the left : this disproportion, however, resulted almost exclusively from hypertrophy of the former, the diameter of which was ascertained to be nine lines. The cavity of the ventricle itself was perhaps slightly encroached upon, but not in a very marked manner, the hypertrophy being rather eccentric than concentric. The fleshy columns were fully, but not abnormally, developed.

On passing my finger into the usual position of the orifice of the pulmonary artery, I was much puzzled at being unable to discover the aperture, and was equally foiled on endeavouring to carry the finger through from the artery into the ventricle. I then carefully cleansed the heart, and discovered the following peculiar and unexpected condition :—The long axis of the ventricle was shortened in the direction of the arterial opening, and, instead of communicating directly with the pulmonary artery, the anterior part of the base of this cavity presented two small circular apertures, neither of which would admit, without distention, the passage of a small-sized goose-quill ; their circumference was dense and white, similar in appearance to the auriculo-ventricular zone. One of these outlets from the ventricle opened into a small but thick muscular sac of oval form, which measured about an inch in its longest diameter, and gave origin to the pulmonary artery. The calibre of this vessel, and the manner of its connection to the small ventricular chamber just described, were normal : it possessed only two semilunar valves, but they were healthy.

Above and slightly to the left of this orifice, and about seven lines distant from it, was the second outlet from the right ventricle, precisely similar to the other in form, charac-

ter and size. This opened into the aorta, or rather into the angular interval between two of its semilunar valves, so that the aperture was partly bounded by the crescentic borders of the proper arterial coat, and in part by the muscular fibres of the ventricle. On examining the canalis arteriosus, this duct was found to be unusually short; and a diaphanous membrane, which had the appearance of a patch of organised lymph with a defined margin in the pulmonary artery, alone obstructed the passage of a probe into this vessel from the aorta.

The systemic arteries (which were injected and dissected throughout the body) were generally of rather small calibre, and presented an unusual number of the more common varieties; such, for instance, as a high division of the brachials, abnormal origin of the obturator and circumflex arteries in the lower extremity, double renal artery on one side, &c. The bronchial arteries were unusually large and tortuous; and the branch from the internal mammary artery which accompanies the phrenic nerve was nearly equal in size to the parent trunk, and expended itself principally in the adjacent adherent lung. The systemic veins presented no peculiarity either in size or distribution. The course and distribution, as well as the calibre, of the pulmonary arteries and veins seemed to be natural, as far as could be ascertained without injection.

A quantity of colourless fibrin occupied a considerable portion of the distended right auricular appendix: it adhered firmly to the walls of this cavity by having insinuated itself between the fleshy columns, which suggested the idea that, *possibly*, it had accumulated gradually during life. It certainly presented more consistence, and was more adherent to the walls of the auricle, than such fibrinous deposits, which are frequently met with as a *post-mortem* appearance, usually are.

In the foregoing case the more prominent symptoms are satisfactorily accounted for by the *post-mortem* appearances; as it is evident that the currents of venous and arterial blood must have been mixed in the aorta, and that the lungs must

have been deprived of a large share of their supply from the right ventricle.

But the sum of the calibre of both the arterial outlets from this cavity was not equivalent to that of the normal aperture of the pulmonary artery; and thence the hypertrophy of the ventricle. To the same cause also may be traced the dilatation of the auricle; for that obstruction which tends to engender increased power in the propelling cavity, from its struggles to overcome the obstacle, must, at the same time, tend to produce distention of the receiving cavity, which is constantly overloaded in consequence of its inability to part with its contents as fast as it is replenished.

The morbid condition of the liver and spleen was doubtless associated with the obstructed venous circulation; but I cannot help regarding it also as indicative of a function performed by one or both of these organs vicariously of the lungs.

It is impossible to overlook the connection between the large size of the bronchial and branches of the internal mammary arteries, and the obstructed pulmonary circulation; —a coincidence which seems to justify the supposition that, naturally, the lungs are not exclusively dependent for their nutrition on the bronchial arteries.

Lastly, as regards the primary cause of all the mischief, it would seem to be a congenital vice of conformation, referrible to that category of cases in which obstruction of the discharging orifice of the ventricle is accompanied by an abnormal aperture for the exit of the blood. I find, however, on reference to authorities on this subject, that in the instances most closely allied to the present, either the ductus arteriosus was open, or the communication was more direct between the ventricles, by perforation of their septum. It is true, in the case I have just narrated, that the abnormal opening was on the ventricular side of the semilunar valves of the aorta, occupying the angular interval between the attachments of two of them; so that, in strict anatomical

language, the communication was really between the two ventricles, as the extent of its middle coat constitutes the limit of the artery. Yet, physiologically considered, the effect both upon the heart and circulation must have been the same as if the blood had been poured into the aorta higher up, or the canalis arteriosus had been pervious: for the left ventricle could have had no additional burden thrown upon it by the communication in question, and the currents of venous and arterial blood must have been effectually mixed.

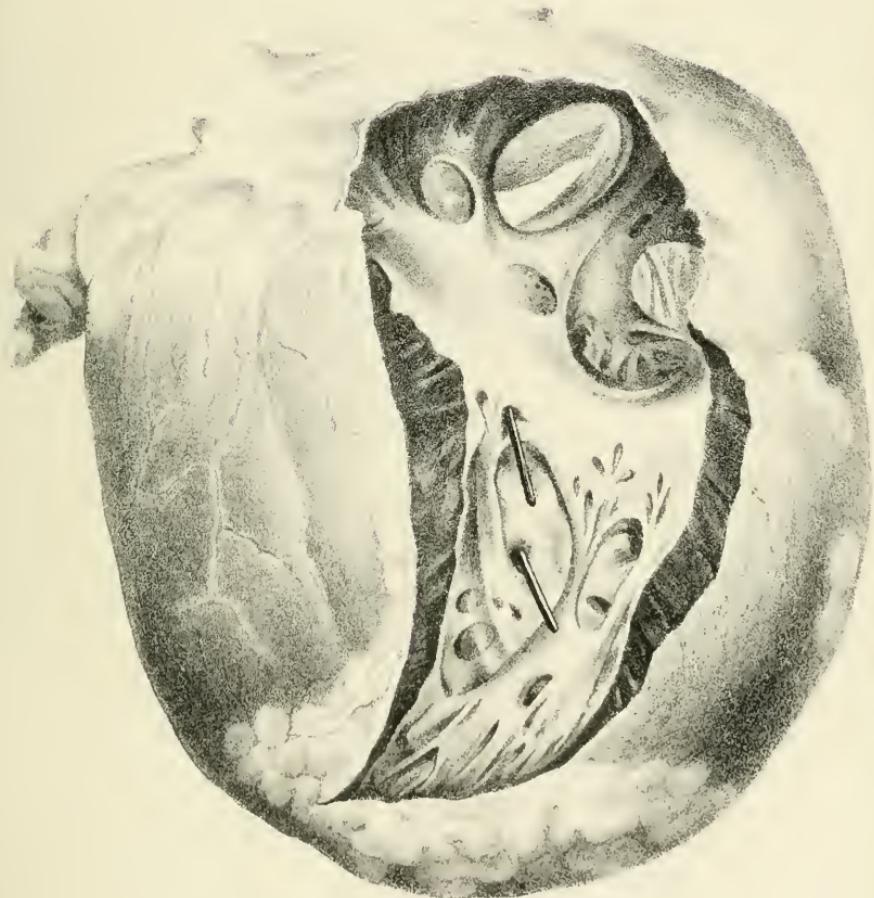
I find, on consulting Otto's Pathological Anatomy, that he refers to instances of malformation belonging to the same class as the present, in which the lungs were supplied by branches from the thoracic aorta, from the phrenic, and from the pericardial arteries. The enlargement of the bronchial arteries in the present case was probably due to the exigencies of the lungs, and illustrative of a general principle where there is an interrupted circulation, rather than belonging to and forming part of the original vice of conformation.

A suggestion was thrown out by some gentlemen who have examined the heart, that the superadded abnormal opening from the right ventricle might have been the consequence of disease. I cannot say I entertain this belief myself, though the *apparently* recent closure of the canalis arteriosus, and the doubt thrown over the early history of the case, might seem to lend some colour to the supposition.

EXPLANATION OF PLATE III.

This plate, illustrative of Mr. Le Gros Clark's case of cyanosis, exhibits the right side of the heart laid open.

The upper aperture, into which a rod is passed, communicates with the aorta; the lower one opens into the small ventricular chamber which gives origin to the pulmonary artery.



OBSERVATIONS
ON THE
CO-EXISTENCE
OF
VARIOLA AND SCARLATINA,
WITH REMARKS ON THE
CO-EXISTENCE OF OTHER ERUPTIVE FEVERS.

By J. F. MARSON,
SURGEON TO THE SMALL-POX AND VACCINATION HOSPITAL, LONDON.

COMMUNICATED BY GEORGE CURSHAM, M.D.

Received March 30th—Read May 26th, 1847.

IN the course of the last eleven years, I have seen, at the Small-Pox and Vaccination Hospital, seven persons who had variola and scarlatina simultaneously. No two of these patients were received from the same place. They all appeared separately, and at various intervals, at times when there was not any other case of scarlet fever in the hospital. The only discoverable disease under which they were labouring, on their arrival, was small-pox; but in the progress of this disease, scarlatina also became evident, between the fourth and the eighth days of the variolous eruption. Judging by the length of time that each disease is known, usually, to remain latent in the system after its reception, before constitutional symptoms are manifested, we may conclude that the germs of scarlatina were received towards the end of the incubative stage of variola, fourteen days being allowed for the development of small-pox, and from five to nine for scarlatina.

It is desirable that it should be clearly understood that these patients had, all of them, the leading symptoms of scarlatina well marked, and that the eruption was different from the roseola which frequently precedes the eruption of small-pox, and also different from the erythema (somewhat resembling it) arising from the miasm of hospitals; in fact, it was the florid red eruption, peculiar to scarlet fever, which can hardly be mistaken for anything else by a medical man, whose eye has once been rendered familiar with it. Three of the patients had small-pox in the unprotected state, and four after vaccination. Six of them were adults, the seventh a child, four years of age. Three were males, and four females. In one it was followed by anasarca, and in two by swelling of the parotid and submaxillary glands, and all had desquamation of the cuticle; a sequence which, it will be remembered, is not usual in small-pox, except in those parts immediately adjoining the pustules, but, on the contrary, almost constant in scarlatina. All the patients recovered but one, and as that case afforded, perhaps, a more striking illustration of the combination of the two diseases than the rest, the particulars are subjoined in full.

Amelia Hayward, four years of age, was admitted into the Small-Pox Hospital in November 1843. She was received on the third day of the small-pox eruption in the semi-confluent form, and had not previously undergone vaccination. She had no dangerous, nor even severe symptoms from the small-pox; but, on the third day from her admission and the fifth of the eruption, there was an evident accession of fever, which continued to increase for two days, when soreness of the throat was noticed, with enlarged tonsils and redness of the skin. The tongue was white in the centre, red at the edges, and elongated red papillæ were perceptible, rising through the white fur; in short, all the leading symptoms of scarlatina were manifest. The redness of the skin continued for four days, when it disappeared; but the parotid and submaxillary glands and surrounding cellular tissue became, about the same time, very much swollen, so that there was

great inconvenience in breathing, owing to the swelling, and an extremely acrid, offensive discharge from the nostrils and ears. She died on the eleventh day after the appearance of the eruption of scarlatina, and the seventeenth of small-pox.

One of the other patients was for some time in great danger from the severity of the two diseases, but ultimately recovered, as did the remaining five.

Domestic servants are often sent, by mistake, during the initiatory fever of small-pox, to the Fever Hospital, at the time when the medical adviser cannot decide, positively, what febrile disease may be approaching. Such cases are generally transferred to the Small-Pox Hospital; but, during the late epidemics, when the hospital was very full of patients, a few of these patients, with the lighter forms of variola, were allowed to continue at the Fever Hospital; and I am permitted by Dr. Goodfellow, then attached to the hospital, to state, that three had also scarlet fever concurrently with small-pox, taken most likely from the scarlet fever patients, near whom they had been placed on first arriving at the hospital.

I may here mention what is only, perhaps, a little remarkable, that three of the patients, with these conjoined diseases, were the servants of medical men. Whether they had, in the execution of their duty, come into contact about the same time with persons labouring under these respective diseases, I cannot say.

It is rare to see two active diseases, such as small-pox and scarlet fever, affecting the body at the same time; as, indeed, it must be, to induce a man of Mr. Hunter's great experience and observation to believe that it never occurred, and to go so far as to state that no two diseases of this kind could go on in the body concurrently. These are his words:—"As I reckon every operation in the body an action, whether universal or partial, it appears to me beyond a doubt, that no two actions can take place in the same constitution, nor in the same part, at one and the same time; the operations of the body are similar, in this respect, to actions or motions in common matter. It naturally results from this principle,

that no two different fevers can exist in the same constitution, nor two local diseases in the same part, at the same time.”* Again:—“In two eruptive diseases, where both are necessarily the consequence of fever, and where both naturally appear after the fever nearly at the same distance of time, it would be impossible for the two to have their respective eruptions even in different parts, because it is impossible that the two preceding fevers should be co-existent.”† He further says:—“But a constitution or part may have equally a susceptibility to a variety of diseases, as venereal, scrofula, &c., some of which may have a common cause, others a specific, as lies; yet the constitution can have, at the same time, only one specific action. As there are susceptibilities for dispositions, so there must be also dispositions for actions; yet two of these cannot exist at the same time in the same part or constitution.”‡ Mr. Hunter states, that the progress of inoculation is suspended if performed at the time when measles is in the constitution, and cites, in confirmation of this opinion, an instance occurring in his own practice. On the disappearance of the measles, the small-pox inoculation went through its usual course, and ended well.§

Unquestionably the examples are but few in which two febrile diseases do occur at the same time, in the same individual; but the rule is not without exception, nor anything like it. Mr. Delagarde,|| of Exeter, gives the particulars of a case in which a person suffered from small-pox and measles simultaneously: and Dr. M'Bride,¶ refers to several such cases as having been seen in Dublin, when measles broke out at the time that a number of children, at the Foundling Hospital, were under inoculation for small-pox. Mr. Leese** relates one such case; Dr. Tracy†† two—one accompanying

* Hunter—Palmer's edition, vol. iii. p. 4.

† Ibidem.

‡ Hunter, vol. i. p. 313.

§ Ibidem.

|| Med.-Chir. Trans., vol. xiii. p. 166.

¶ Practice of Physic, vol. ii. p. 112.

** Medical and Physical Journal, vol. iv. p. 29.

†† Ibidem. vol. iii. p. 572.

inoculation, the other occurring with natural small-pox. Two are given in the report of the New Town Dispensary, Edinburgh.* "In one, the eruption of measles came out on the fifth day of the eruption of small-pox, and both ran their usual course. In the other, the eruption of small-pox came out on the third day of the eruption of measles, which continued visible for two days more." Other cases may be found reported in the *Gazette des Hôpitaux*,† from the *Hôpital des Enfants Malades*, Paris.

An instance of vaccine disease and measles existing contemporaneously, without interfering with each other, is accurately given by Mr. Gilder‡ of the Coldstream Guards; and many correspondents of the periodicals,§ and I also, have several times seen small-pox and the vaccine disease advancing *pari passu*, without the usual progress of each disease, respectively, having been interrupted, although generally the vaccinia is checked immediately on the outbreak of variola.

Measles and scarlet fever were observed together frequently, by the French, many years ago,|| and more recently by M. Guersent,¶ at the *Hôpital des Enfants*, Paris.

There is a case on record of varicella and vaccinia.**

Hooping cough, I may, perhaps, be allowed to include in the list, although it is not one of the eruptive fevers; and therefore not strictly within the limits I have assigned to myself in this communication. It is, however, like them, essentially infectious and inflammatory in its character, a disease of great danger, often leading to fatal results. Dr. Willan†† has seen it in conjunction with variola; and cases of hooping cough and measles are mentioned, in the report

* Edinburgh Medical and Surgical Journal, vol. xv. p. 314.

† Tom. viii. p. 34. 1834.

‡ Med.-Chir. Trans., vol. xii. p. 186.

§ English Medical Journals, *passim*.

|| Recueil Periodique de la Société de Médecine de Paris, tom. viii. p. 359.

¶ Gaz. des Hôpitaux, 1834, tom. viii. p. 34.

** Lond. Med. Gaz., vol. ii. p. 633.

†† Reports of Diseases of London, p. 38.

from the Dispensary for the Diseases of Children, Doctors' Commons,* as having been frequently seen at once in the same individual. Three cases of this combination were observed by Dr. Robt. Williams :† "they all occurred in the same family in the depth of a severe winter, and all proved fatal."

Some years ago a statement was in circulation that vaccination would cure hooping cough, founded I suppose on the axiom of Hunter, that no two febrile diseases could go on at the same time in the same constitution ; but, so far as I have known, this statement requires some qualification. Hooping cough and the vaccine disease may often be seen co-existing, each pursuing its course without interfering or being interfered with by the other. The same law appears to operate here as in other like instances, one disease interrupting, occasionally, the progress of the other ; but it is of less general application than when the two diseases have been received into the constitution casually. However, as the proceeding does no harm, there is not any objection to its being practised whenever opportunity offers : the vaccination is not rendered less effectual, that I am aware of; and as the tendency of these active diseases is, frequently, for one to suspend the action of the other, it will, doubtless, succeed on some occasions. The vaccination usually takes well.

There is a case reported in the *Lancet*,‡ by Dr. Barnes, of the presumed co-existence of small-pox and scarlatina, and the statement is rendered very likely to be correct from the two diseases being at that time in the house ; but, from a careful perusal of the details of the case, I am disposed to think it was one of those instances of small-pox preceded by roseola, not unfrequently seen after vaccination.§

Dr. Gregory|| says, in an article written before the period

* London Med. Repos., vol. xiii. p. 90.

† On Morbid Poisons, p. 303.

‡ Vol. i. 1845, p. 610.

§ See a similar case in the British and Foreign Med. Review, vol. iv. p. 219, translated from a foreign Journal.

|| Cyclop. of Pract. Med., vol. iii. p. 744.

embraced in this paper, "it has occurred to us to see at the Small-Pox Hospital several unequivocal cases of the simultaneous existence of small-pox and scarlatina anginosa;" and Dr. R. Williams* observes that "the variolous poison is capable of co-existing with many other poisons; also of influencing their actions, and of being reciprocally influenced by them. Dessesarz has seen variolæ co-exist with scarlatina, and with the hooping cough."

Mr. Wilson† remarks, "variola is occasionally complicated with rubeola and scarlatina, and sometimes with petechiæ."

In the *Lancet*‡ a case of small-pox and scarlatina will be found translated from Stift's *Medical Annual of the Austrian States*.

"Ring even mentions a case of triple disease co-existing, or of the small-pox, the measles, and the hooping cough, all of which ran their course together."§

Roseola and erysipelas might also be mentioned as being occasionally blended with small-pox, but that I consider the roseola which often precedes the eruption of variola as part of the disease itself; and I regard the erysipelas superinduced by patients being placed in a vitiated atmosphere, as a result which we can almost certainly bring about at will, just as readily as we can insure the effect of any other known poison: and although we recognise erysipelas, when taking place alone, as a special disease, we hesitate to do so when it is complicated with another. A great deal might, doubtless, be said in favour of its individuality. Like other diseases, it is often the result of an unknown cause; on the other hand, it is also often induced by causes that are well known,—or, at least, we know the combination of causes that will induce it,—but it is not the less a special disease on that account; the other febrile eruptive diseases, which we call specific, being the result of unknown causes. Erysipelas is excited by a

* On *Morbid Poisons*, vol. i. p. 211.

† On *Diseases of the Skin*, p. 84.

‡ Vol. ii. 1834-5, p. 446.

§ Dr. R. Williams on *Morbid Poisons*, vol. i. p. 211.

poison that may fairly be called a specific one ; others, giving rise to the eruptive diseases, we call specific poisons, but we know not what they are, except by name and in their effects. It bears a very strong analogy to the other eruptive fevers, all of them most likely having had their origin in occult morbid animal poisons, derived either from man or the lower animals, as that has ; and it is, like them, capable of being propagated by inoculation ; the sponges of an hospital, for instance, conveying it often from one patient to another, thus showing its likeness, in this respect, to small-pox, cow-pox, measles, varicella, syphilis, plague, farcy, &c., all of which can be imparted by inoculation to other individuals ; and I have but little doubt that scarlet fever might be communicated in the same way. Two of these diseases, namely, cow-pox and farcy, we believe, nay are almost certain, originated with the lower animals ; and small-pox is traditionally reported to have had its rise from the camel.

Whenever erysipelas comes under our notice in combination with other diseases, we may strongly suspect, if not feel certain, that there is defective ventilation or some other mismanagement in the apartment of the patient, even when it occurs in private houses, as it sometimes does ; for it should never be forgotten that the presence of one patient in a small room may, by neglect of proper ventilation and cleanliness, produce a condition of the air quite as impure as that in the larger space of a crowded hospital. This state of things I have known to take place in private houses more than once.

I have referred to most of our best systematic works on disease, as well as to the majority of our periodicals published during the last thirty years, but do not find allusion made, in any of them, to the co-existence of these two diseases, small-pox and scarlatina ; and I am not aware that any British writer, with the exception of those I have quoted, has noticed the circumstance of their ever occurring together. The French, however, are no strangers to the fact. In the *Dictionnaire de Médecine*, Art. *Variole*, the writer says, under the head of "*Varioles Compliquées*," "La variole peut être

compliquée accidentellement avec la rougeole, la scarlatine, et plus souvent avec le purpura hæmorrhagica." Rayer* also remarks, that small-pox may be complicated with measles, scarlatina, purpura, croup, pneumonia, &c. ; and I find that as far back as the seventh year of the République Française (1799), three cases of concurrent variola and scarlatina are reported as having been observed by M. G. Vieusseux.†

A case is given at length in the *Gazette Médicale de Paris*,‡ from the Italian journals, by M. Spadafora. Another by M. Baudelocque ;§ and others may be found scattered over the French periodicals during the last few years.||

It may be said that the history of these rare cases, for such they must be considered, is not of much use in practice ; but as the two diseases have occasionally been seen associated, it appears to me to be desirable that the possibility of such a recurrence should be generally acknowledged.

Thus, either from personal observation or from the writings of others, I present examples of the simultaneous occurrence of variola and scarlatina, variola and rubeola, variola and pertussis, variola and vaccinia, rubeola and scarlatina, rubeola and vaccinia, rubeola and pertussis, varicella and vaccinia, pertussis and vaccinia.

It has most likely occurred to some of the Fellows of the Medical and Chirurgical Society to witness instances analogous to those I have adduced.

* Art. *Variole*, 2^{de} édition.

† *Recueil Periodique de la Société de Méd. de Paris*, t. 6, p. 417.

‡ 1837, p. 234.

§ *Gazette des Hôpitaux*, 1832, t. 4, p. 74.

|| *Gazette des Hôpitaux*, 1832, t. 5, p. 411. *Gaz. Méd. de Paris*, 1834, p. 312.

CASE OF
MALFORMATION OF THE HEART,

IN WHICH

DEATH RESULTED FROM OBSTRUCTION IN THE TRUNK
OF THE PULMONARY ARTERY.

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Received April 7th—Read May 25th, 1847.

THE following case is conceived to be sufficiently interesting, from the unusual form of the malformation of the heart and the infrequency of the cause which occasioned death, to render it worthy the notice of the Society.

William Holland, a milk boy, aged 15, was admitted into the Royal Free Hospital, on the 20th of February 1847. He stated that on the 12th he had bruised the left knee by a fall, and had since suffered from constant pain in that joint, and also, for two or three days before admission, in the right knee. The day after the accident he began to experience pain in the left side of the chest and difficulty of breathing.

When admitted he was much collapsed and the extremities were cold and livid. The cheeks were of a deep purple colour and the lips blue. The fingers and toes were club-shaped, and the nails incurvated and very dark-coloured. The pulse was 124 in the minute and extremely feeble. The tongue dry and covered with a whitish fur. The respiration

was peculiarly rapid and panting, and he was compelled to lie on the back, partly inclined towards the right side, and with his head low. He complained of pain in the region of the heart, palpitation, and difficulty of breathing. Both knee-joints were swollen and tender, and there existed a red and swollen patch over the left trochanter.

The chest yielded a clear sound on percussion, except in the praecordial region, where the resonance was impaired over a larger space than natural. The liver could be felt extending a little below the edges of the ribs. The sternum was arched and prominent, more especially towards its base. The respiration was puerile in its character, and was attended with occasional mucous rales. A loud murmur was heard in the praecordia, accompanying the impulse of the heart. It was of a soft or blowing character, and was most intense at the cartilage of the third left rib near the sternum, or at a point half an inch above the nipple and between that body and the sternum ; it was here prolonged throughout the whole period, so as to render the second sound inaudible. From this point it continued to be heard very distinctly, though decreasing in intensity, along the upper part of the sternum, in the subclavian and carotid arteries, and on the left of the spine in the interscapular and dorsal regions. It was also heard less distinctly in a line from between the nipple and sternum, towards the middle of the left clavicle. Below the level of the nipple the murmur became shorter and more feeble ; and, at the point of pulsation of the apex, towards the epigastrium and on the right side of the lower half of the sternum, it was followed by a very clear second sound.

The boy was much exhausted, and his intelligence so impaired that it was found impossible to collect any satisfactory information of his state of health previous to the present attack. He stated, however, that he had lost flesh and strength ; and had been very subject to affections of the chest, and of a livid complexion, since he was thrown from a cart twelve months before.

These notes were taken about 4 P.M. on the day of ad-

mission ; and, notwithstanding the free use of stimulants externally and internally, he gradually sank, and died at eight o'clock on the following morning. On inquiry I ascertained that he had always been of a somewhat livid complexion ; but was stout, healthy, and capable of a full amount of exertion, till the occurrence of the accident he referred to. He was then riding at the back of a cart, when it toppled up, and he was thrown out and fell upon his head. He was admitted into the hospital on the 6th of February 1846, under the care of my colleague, Mr. Gay, with symptoms of concussion of the brain. He continued there six days, and the only peculiarity then observed in his appearance, was some slight lividity of the lips. Since that time he had been gradually getting thinner and weaker ; he was constantly chilly, and very subject to take cold. He complained occasionally of palpitation, difficulty of breathing, and pain in the region of the heart ; and his hands and face were always very blue, but especially so in cold weather, or when he was suffering from affection of the chest. His appetite was generally defective, and he occasionally vomited his food. His father is of a livid complexion and has a "pigeon breast."

The *post-mortem* examination took place at 4 P.M., on the 22nd, thirty-two hours after death.

The brain was healthy, though much congested. It weighed 49 oz. 4 dr. avoirdupois.

The surfaces of the pleura on the right side were adherent by a small cellular band. The left lung was entirely free. Both lungs were engorged with blood, sparingly crepitant, and contained several masses in the state of pulmonary apoplexy. The bronchial mucous membrane was somewhat reddened. The pericardium was free from adhesions, and did not contain fluid. The heart was about 10 oz. in weight. It was broader from side to side than from above downwards. Its total circumference was $8\frac{1}{2}$ inches, of which the right ventricle constituted $4\frac{1}{2}$.

The systemic veins were free from disease. The right

auricle was large and its walls thick. The foramen ovale was closed, with the exception of a valvular aperture capable of admitting a goose-quill. The Eustachian valve was of moderate size. The right auriculo-ventricular aperture measured 39 lines in circumference. The valves were free from thickening and competent. The muscular column to which the cords of the anterior fold were attached was very large and firm. The aperture opened as usual into the sinus of the right ventricle; but this portion of the cavity was separated from the infundibular part by a thick muscular septum, defective only at its centre, over a space of sufficient size to admit the fore-finger, and perforated by one or two very small pores nearer the apex. The cavity thus formed communicated with the aorta by an orifice 30 lines in circumference, situated at its posterior extremity. Its walls averaged $5\frac{1}{2}$ lines in thickness, and were unusually firm and solid. In places they had undergone the fibro-cartilaginous degeneration throughout their whole extent, and the corresponding external serous envelope was opaque. The second or infundibular cavity of the ventricle was of smaller capacity than the former, and gave origin, as usual, to the pulmonary artery. Its walls averaged only 2 to 3 lines in thickness. The orifice of the pulmonary artery was very small, and was provided with only two valves which were extremely thick and opaque. The valves projected into the cavity of the vessel, so as to leave a deep sac behind each, and by their free borders occasioned a further contraction of the arterial orifice; which, while on the ventricular side it admitted a ball measuring 15 lines in circumference, could only give passage to one of 13 lines. The coats of the pulmonary artery were much indurated and thickened, and its canal was entirely obstructed by fibrinous coagula. At the sides of the vessel these coagula were of a dirty white colour, and were laminated and firmly adherent to the valves and diseased lining membrane; but towards its centre they were softer and less decolorized. The obstruction occupied the whole trunk of the vessel and extended a few lines into each division. The distal branches were free

from disease. The ductus arteriosus was impervious throughout the largest portion of its extent, but displayed a conical cavity, extending 2 or 3 lines from the bifurcation of the pulmonary artery. The pulmonary veins were natural. The left auricle was small and its lining membrane opaque. The auriculo-ventricular valves were healthy, and the aperture measured 36 lines in circumference. The left ventricle was of small capacity; its walls felt flaccid, and were $3\frac{1}{2}$ to $4\frac{1}{2}$ lines thick. The aorta communicated with this ventricle by an orifice of about the same size as that by which it arose from the right ventricle.

The aorta was very large from its origin to the sulus marking the former point of communication with the ductus arteriosus. From that part its calibre greatly decreased. Its valves were three in number. They were free from disease, and completely closed the orifice. The coats of the vessel were natural. The bronchial and oesophageal branches were somewhat large.

The large veins and the several cardiac cavities were much distended with blood, chiefly fluid or feebly coagulated. The abdominal organs displayed no signs of disease, but were much engorged. The liver weighed 45 oz., the spleen $6\frac{1}{2}$ oz., the left kidney $4\frac{1}{2}$ oz., and the right $3\frac{1}{2}$ oz. The latter was somewhat mottled.

Remarks.—The form of malformation of the heart in which the pulmonary orifice is contracted and the ventricular septum deficient, is not of uncommon occurrence; the combination, however, of a supernumerary septum in the cavity of the right ventricle with this malformation has, so far as I am aware, been very rarely observed.

When congenital perforations exist in the inter-ventricular septum, they are situated at that portion of its base which remains throughout life unprotected by muscular substance; and form communications between the origin of the aorta and the sinus of the right ventricle. When these communications are free and direct, the upper portion of the sinus of the right ventricle is usually found more or less distinctly sepa-

rated from the infundibular portion and origin of the pulmonary artery, by the column of muscle reflected from the pulmonary orifice along the upper and anterior border of the ventricle. A case well illustrating this condition was recently described by myself in the *Monthly Journal of Medical Science*.* In the present instance, however, the muscular mass referred to is of unusual size, and is combined with great enlargement of the fleshy column to which the anterior fold of the tricuspid valve is attached, and general hypertrophy of the walls of the ventricle, so as to produce a nearly complete septum in the centre of the cavity. Of cases of malformation similar to that now described I have only been able to collate four or five examples. Of these, one is reported by Mr. Holmstead;† a second, of which the preparation exists in the Museum of St. Bartholomew's Hospital, is briefly described by Dr. Farre;‡ and a third, bearing, however, a less close analogy to the present case, is related by Dr. Crampton, and subsequently by Dr. Todd.§ Dr. Theophilus Thompson has recorded a case in some respects similar;|| and a fifth has very recently been communicated to the

* March 1847, p. 644, vol. vii., or *N. S.* vol. i.

† *Medical and Physical Journal*, vol. xvii. p. 455, 1822. In a girl who died at the age of 9 years. The description is accompanied by a woodcut, showing the aorta arising from both ventricles, and the aperture in the supernumerary septum. The pulmonary artery is stated to have been naturally formed in this case. The foramen ovale was closed.

‡ *Malformations of the Heart*, p. 26. The age of the person is supposed to have been about 14, but there is no history of the symptoms during life. The malformation is very similar to that in the present case, with the exception that the pulmonary artery, though of small calibre, has three well-formed semilunar valves at its orifice. The foramen ovale appears to be impervious.

§ Crampton, *Trans. of Dublin College of Physicians*, *N. S.* 1830, vol. i.; and Todd, *Cyclop. of Anatomy and Physiology*, vol. i. p. 614. The pulmonary artery was in this case destitute of valves, but there existed a puckering at its orifice which occasioned contraction. The foramen ovale was open, and the septum of the ventricles imperfect. The heart was that of a boy aged 10 years.

|| *Med.-Chir. Trans.*, vol. xxv., 1842, p. 247. In this instance the su-

Society by Mr. Le Gros Clark.* In all these cases the supernumerary septa were situated in the right ventricle, at or near the same point, and co-existed with some form of defective development at the orifice of the pulmonary artery ; and, with the single exception of the case of Dr. Thompson, with an imperfection of the septum ventriculorum, so that the aorta communicated in part with the right ventricle. In three of the cases the irregular development might, as suggested by Dr. Todd, be referred to the obstruction at the pulmonary orifice, occasioning dilatation of the infundibular portion of the ventricle and the general hypertrophy of the walls of the cavity, and more especially of the fleshy columns forming the septum in its centre. This explanation is, however, inapplicable to the cases of Mr. Holmstead, Mr. Le Gros Clark, and Dr. Thompson, in which no contraction of the pulmonary orifice existed.

It is evident that this form of malformation must greatly interfere with the passage of the venous blood into the pulmonary artery, and, to an equal degree, favour its entrance into the aorta. In the case now related, after the occlusion of the ductus arteriosus, certainly not half the column of blood returned to the right auricle can have been transmitted to the lungs ; yet the degree of cyanosis, till within a year of the boy's death, bore no just relation to this free intermixture of the currents of blood.† During this period, it is probable that

pernumerary septum co-existed with an excess in the number of pulmonary semilunar valves, there being four of equal size and well formed. The pulmonary orifice was one inch wider than that of the aorta. In the septum there was an aperture one inch in length, and half an inch wide. The patient, a female, 38 years of age, was healthy till she had an attack of the oriental cholera ; her symptoms were aggravated two years before death.

* See page 113. Supernumerary septa are mentioned as occurring in the heart's cavities both by Andral and Rokitansky, but without any case of the kind being described. The latter states that they occur in both ventricles.

† In Mr. Holmstead's case, though the proportion of venous blood entering the general circulation was scarcely less than in the present instance, the child had no signs of any cardiac affection, and was, indeed, of a re-

the pulmonary artery was free from disease, and its defective capacity being compensated by the direct passage of the blood from the right ventricle into the aorta, no material disturbance of the balance of the circulation was occasioned. At the time of the accident, however, the pulmonary artery seems to have sustained some injury, which gave rise to the disease of its coats, and a further contraction of its canal; the communication between the right ventricle and aorta became no longer equal to the transmission of the column of blood required to compensate the decreasing circulation through the pulmonary artery; the right ventricle was imperfectly emptied of its contents; the congestion gradually extended throughout the venous system, and the lividity of the face and extremities became proportionately more marked. This view of the case tends to confirm the explanation of the cause of cyanosis, first suggested by Morgagni, and since more fully developed by Louis.

The observations of Baron* and Paget† have attracted attention to the formation of coagula in the pulmonary artery as a cause of death; so far, however, as I am aware, in the cases of this description observed, the coagula have been found in the branches of the pulmonary artery; and I have not met with any recorded instance of primary obstruction of the trunk of that vessel.‡ In this case, the diseased condition of the arterial tunics, the complete decolorization of the outer layers of coagulum, and their firm adhesion to the lining membrane of the vessel, render it most probable that the obstruction had been in progress for a considerable period, without having occasioned any urgent symptoms.

markably healthy appearance up to the age of three and a half years, when cyanosis supervened on an attack apparently of inflammation of the lungs.

* Arch. Gen. de Med. III^{me} et nouvelle série, t. ii. 1828, p. 6.

† Med.-Chir. Trans., vol. xxvii., 1844, p. 162; and vol. xxviii., 1845, p. 353.

‡ The case of M. Hélin, quoted by M. Baron, from the Bulletin de la Société Anatomique, Oct. 1837, may possibly be a case of primary obstruction of the trunk of the pulmonary artery. Arch. Gen. 1828, p. 22.

The final closure of the canal of the vessel appears, however, to have been a more acute process, and to have commenced coincidently with the rheumatic affection following the fall, eight days before death. In the drawing and preparation, the obstruction in the pulmonary artery appears less complete than it really was, owing to several masses of lymph, adherent to the valves and filling the spaces behind them, having been removed, to expose and render more evident the faulty conformation of that part of the vessel. Mr. Paget has remarked, in reference to the sixth case related in his first valuable memoir on obstructions of the branches of the pulmonary artery,* that when only two valves have been found at the pulmonic or aortic orifices, those valves have very generally been the seat of disease. The present affords a further illustration of this fact; and I have, in several instances of defect in the number of valves at the aortic orifice, seen their folds extensively diseased. The case related by Mr. Paget presents, in reference to the state of the pulmonary artery, considerable resemblance to that here described.

The physical signs which have been reported as present in this case, corresponded very closely with those observed in the other instance of somewhat similar malformation of the heart to which I have alluded.† In both, a loud murmur accompanied the systole of the ventricle, and was most intense at the seat of the aortic orifice. It was of a soft or blowing character, and was very distinctly audible, though gradually decreasing in intensity, throughout the course of the thoracic aorta. At the base of the heart, it was so prolonged as entirely to mask the second sound; but below that point, at the seat of pulsation of the apex, towards the epigastrium, and on the right side of the sternum, the murmur was feebler and shorter, and was succeeded by a second

* Med.-Chir. Trans., vol. xxvii. p. 182.

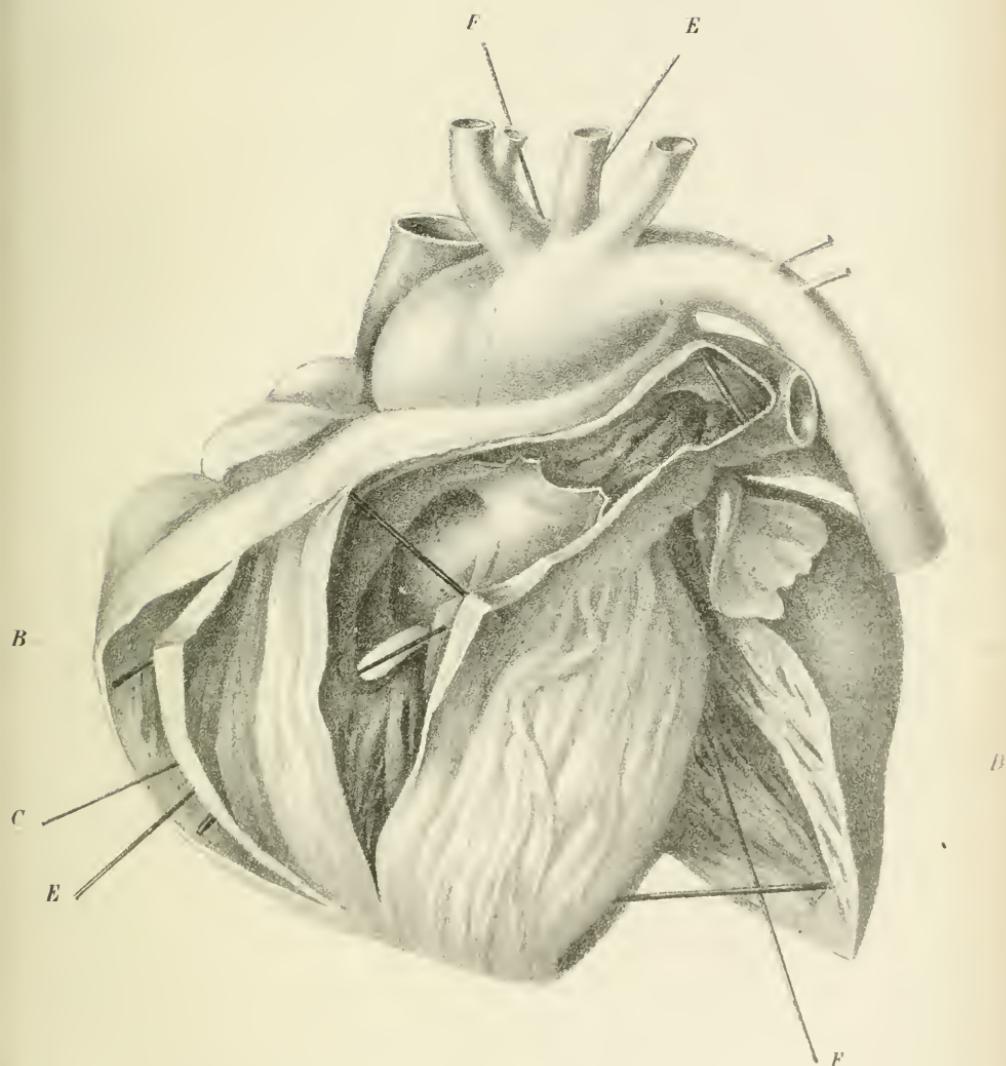
† They will also be observed to be similar to those assigned by Dr. Hope, partly on theoretic grounds, as characteristic of these forms of malformation.

sound of a peculiarly ringing or flapping character. It was also audible in a line from midway between the sternum and left nipple, towards the middle of the clavicle, though it was less intense here than along the upper part of the sternum. From these observations it may be inferred, that the murmur was chiefly occasioned by the meeting in the aorta of the two currents of blood proceeding from the right and left ventricle, and also, in part, by the constriction at the orifice of the pulmonary artery. The peculiarly ringing or flapping character of the diastolic sound is, doubtless, referrible to the powerful reaction of the large column of blood in the ascending aorta, on the semilunar valves at its origin. I have at present under my care a child about five years of age, in whom the physical signs are precisely similar to those here related; and these, together with the symptoms of obstruction at the right side of the heart which have been present from early life, have led me to infer the existence of similar malformation.

EXPLANATION OF PLATE IV.

This plate illustrates Dr. Peacock's case of Malformation of the Heart.

- a, Infundibular portion of the right ventricle, with the orifice and trunk of the pulmonary artery laid open.
- b, Portion of the anterior wall of the sinus of the right ventricle, turned back so as to expose the cavity.
- c, Probe introduced into the opening in the septum, by which the two portions of the right ventricle are divided.
- d, Cavity of the left ventricle laid open.
- ee' ff', Probes passed from an opening in the posterior side of the aorta into the cavities of the right and left ventricles.



ON

SUB-ACUTE INFLAMMATION OF THE KIDNEY.

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IT is not my intention in the present paper to enter into unnecessary details respecting a disease, with the general features of which it may be presumed that all members of this learned Society are sufficiently conversant. Merely glancing at such portions of the subject as are already familiar to the Profession, I shall endeavour rather to fix attention on changes hitherto little observed; changes which commence in inflammation of the kidney, and tend completely to disorganise it, but which run a slow and insidious progress during life, and often leave in the dead body no such obvious traces as would strike the superficial observer.

The latent processes to which I refer are among the most interesting and curious in the whole compass of pathology; consisting (1) in the more or less complete obliteration of all that is characteristic in the ultimate structure of the kidney; and (2) in the attempt to compensate for this, by the organization of a new secretory apparatus.

An examination of this latter process,—by which (as I shall presently show) vesicles are substituted for the normal tubularity of the gland,—will lead me to explain the origin of

those large renal cysts, so well known in the dead-house ; but first, in order to elucidate that earlier stage of the disease, in which the tubes and Malpighian tufts are destroyed, I shall attempt to define more exactly the nature of nephritis, and shall describe those primary lesions of structure which attend it. Such observations as I have to offer on these topics will be rendered clearer, if I may be allowed to preface them by a few words on glandular inflammation generally.

Many pathologists have inclined to conceive inflammation in glands as in a great degree foreign to the true apparatus of secretion ; they have thought it essential that products of the inflammatory process should have been deposited interstitially to the secreting elements, and should have remained palpable in the rough structure of the organ ; and they would hesitate to use the name of "chronic inflammation" for any other disease than one, where, as in cirrhosis, they might find a hardness to handle, or might point to the supposed maturity of a previous inflammatory infiltration. This view of the case, though substantially correct in respect of the intenser forms of inflammatory action, is too limited and absolute ; for it would probably exclude the entire class of sub-acute glandular inflammations. Organs may, I conceive, rightly be said to suffer inflammation, whenever their natural functions are deranged by an excess of nutrition ; the test of that excess being, that their supply goes beyond their capacity of growth or of secretion. Thus, while we withhold the name of inflammation from the increased nutrition of the pregnant uterus, or of the lactating breast, or of the diabetic kidney, because such increase is commensurate with the necessity, and applied to the purposes, of growth or secretion, we *do* give that name to the case where a superfluous blastema becomes commingled with the secretion, as in active albuminuria, or undergoes interstitial development, as in cirrhosis of the liver ; and in both these instances with equal pathological accuracy.

Inflammations of simple mucous membrane mainly evince themselves in derangement of the secretory functions ; and,

especially if sub-acute, by no means necessarily imply any sub-mucous deposit of organisable material. The modified secretion of such a surface may be eliminated with a rapidity that renders accumulation impossible ; and then (as in gleet or in ophthalmia) how little, beyond a doubtful hyperæmia, would remain for the morbid anatomist !

All this is notoriously true in respect of mucous membranes : it is equally true, though less notorious, in respect of the true glands, which are but processes or involutions of the same structure. No interstitial effusion of lymph need exist in a gland to warrant us in accounting it inflamed ; its inflammation may consist simply in functional derangement, and, during life, may be recognised only by admixing its albuminous products with those of normal secretion.

There is, however, a momentous difference in the two cases. The simple mucous membrane sheds from its surface whatever inflammatory secretions may have formed there ; it is quit of them : they cannot re-act on it injuriously. But the glands have not the same facilities for eliminating morbid products : in proportion to their complexity of structure, and to the narrowness of their canals, they must be liable to embarrassment from changes in the physical qualities of their own secretions ; the delay, accumulation, and obstructive action of which would (it is obvious) suffice to derange the whole microcosm of the organ in which it might occur : and thus an amount of irritation, which on a simple mucous membrane would be utterly insignificant, may, if transferred to a gland, serve to originate its complete disorganisation.

These preliminary remarks will not, I hope, appear to the Society otherwise than appropriate. The influences just alluded to are precisely those which most of all illustrate my subject ; for it is under their operation in the kidney, that the lowest amount of inflammatory action, or even an excitement of function hardly rising to inflammation, may and does, by its secondary and reactive results, become a thoroughly irreparable disease.

Without dwelling on those excessively rare cases, where

idiopathic nephritis (independent of tubercles or of calculus) may, by its mere intensity, have ended in large suppuration or (almost uniquely) in gangrene, I may state that, in an infinite majority of instances, inflammation of the kidneys is sub-acute. It depends on some humoral derangement of the entire system, and commences as functional excitement manifested in an act of over-secretion. The morbid material which thus stimulates the kidney in its struggle for elimination will sometimes consist of products of faulty digestion—the lithates or the oxalates; sometimes of matters cast upon the kidney in consequence of suppressed function in other organs—the skin, or the liver; sometimes will be the mysterious ferment of a fever poison*—typhus, or scarlatina. In these several cases, whatever variety may exist in the detail of their causation, the essential symptoms during life and the essential anatomical changes are strictly identical in kind. They vary only in degree. The *materies morbi* seeks to effect its discharge by means of an increased activity in the secreting functions of the kidney; it stimulates it; and the result of the stimulation is not so much an increase of the watery secretion as it is an augmented cell-growth in the tubules of the gland.† This acceleration of function is incompatible with maturity of the secreted products; the epithelial cells undergo various arrests or modifications of development, and become more or less palpably imbued with evidences of inflammation.

If attention happen to be directed to the state of the urine, that fluid will be found to present manifest signs of derange-

* Perhaps it is not sufficiently known that scarlatina is by no means the only febrile blood-disease which affects the kidney. It is one of many. Béquerel and Franz Simon have frequently found the urine albuminous in cases of small-pox, typhus, and erysipelas. In the first two of these diseases I have repeatedly seen just the same condition of urine as one finds in scarlatina,—the same epithelial and fibrinous casts, &c.; and in the dead body I have noticed the same structural lesions.

† It was conjointly with Dr. Johnson, in the Autumn of 1845, that I made my earliest observations of this peculiar activity. Our joint examinations were referred to in his Paper of that date.

ment. Microscopical examination will show in it numerous nucleated cells, which, in the hurry of over-secretion, have descended from the urinary tubules. Many free cytoplasmic cells will likewise generally present themselves, together with a variety of those indefinite shapes, which are known to the morphologist as abortions of cell-growth, and which constitute a series of connecting forms between the pus-globule and the healthy gland-cell. Mingled with these, in greater or less quantity, will be noticed also those remarkable fibrinous threads first described by Dr. Franz Simon in connection with renal disease. They are seen as exceedingly delicate, almost perfectly transparent and colourless cylinders, often containing in their mass some of the cell-forms just enumerated, or, not unusually, a few blood-discs, resulting from haemorrhage into the tubules.

On several occasions, where the renal irritation has been gouty, I have seen crystals of lithic acid thus entangled in fibrin: in other cases, though far less frequently, I have distinguished crystals of oxalate of lime similarly enveloped. It is well known that these little cylinders are fibrinous moulds of the inflamed urinary tubules, some of the other contents of which they bring with them in their descent. They are thus quite as characteristic of the disease they attend as croupy expectoration is of tracheitis; and the cells or crystals included in them often afford the most valuable therapeutical indications.

Of course, under all the circumstances last mentioned, the urine contains albumen and will yield a precipitate with nitric acid; but, so much more delicate is the microscopical than the chemical test, that it has twice happened to me lately, in cases of scarlatina, to find groups of blood-corpuses in the urine when nitric acid had given no precipitate visible to the naked eye.

While the urine is presenting these characters, there co-exists, with such signs of renal disturbance, a remarkable tendency to serous effusion in various parts of the body; and frequently the startling occurrence of inflammatory dropsy

will first draw attention to the primary mischief. The explanation of this concurrence is easy and unquestionable.

If patients chance to die while their urine is first furnishing the signs enumerated, it will often happen that the kidneys in their general appearance present no marked deviation from healthiness. Their cortical substance may indeed show the minute blood-dots of intra-tubular haemorrhage; or, more rarely, may present here and there a pin-head abscess. But often, perhaps most often, a superficial observer would pronounce the kidneys healthy, and, unless previous knowledge of the albuminuria had existed, they would receive no further attention: or the case-book might contain that vaguest of all vague records—"slight congestion of the kidney."

On minuter analysis, however, the microscope will reveal a large amount of disease. The ultimate tubules are found, as one might anticipate, gorged with an uneliminable excess of crude and vitiated secretion. Blood, and amorphous matter, and an infinite range of cell-growth, from pus-globules to the healthy germination of the gland, present themselves in various combinations; and among them, shape or colour will sometimes enable us to discern the specific cause of the derangement—crystals of lithic acid or of oxalate of lime, or the ochreous tinting of bile. By products such as these the tubes are plugged, irregularly distended, and not unfrequently burst and annihilated. So close is the compaction of material, even in many of those tubes that have no shaped inflammatory products within them, that they are plainly impervious; and it is only by artificial means—by further tearing of the fragment, or by use of chemical agents, that we can satisfy ourselves that the dense plug in question consists but of agglomerated gland-cells.

I have never seen the disease, in the form under consideration, otherwise than double. It varies, of course, materially in respect of intensity, and sometimes will appear to affect with its utmost severity every portion of both kidneys. How gravely it must interfere with the secretion of urine (particu-

larly of its watery part) will be obvious on the slightest reflection, and will sufficiently explain its tendency, so soon as it has made some way, to induce serous effusions, and thus to originate the disease known as inflammatory dropsy.

It is impossible to doubt that many unexplained deaths must have depended on structural changes such as these: the morbid action un-recognised during life, and its traces overlooked afterwards. But, unless the inflammatory process has been universal in both kidneys, and of sufficient intensity to involve the almost complete obliteration of glandular substance, it more usually happens that the patient survives a first invasion of the disease, and his albuminuria becomes chronic, or, under the most favourable circumstances, vanishes.

It can very rarely happen that a patient will emerge from the perils of acute albuminuria, with his kidneys quite free from permanent injury: a certain proportion—perhaps a very small one, but almost certainly some—of the tubular structure will have perished. The former function of this obliterated part has now to be distributed, and discharged vicariously by the rest of the organ. If happily the spoiled tubules are few, their surviving fellows are of course abundantly competent to perform the slight increase of task divided amongst them, and the patient enjoys a complete recovery. But if, on the contrary, the minute structural lesions which I have described, have been diffused in an aggravated form throughout a large portion of both kidneys, so that a great destruction of tubular substance must have occurred,—then it is not so easy for the residual sound texture to compensate for what has perished: it is “working double tides” or something more: it can only do this at its own expense: it finds it a hardship, an inconvenience, an injury. It is under these circumstances, probably, that the symptom of albuminuria becomes chronic. Its persistence depends, no doubt, on the habitual super-stimulation entailed on the surviving structure. The continued over-work is in fact a chronic inflammation.

Under judicious treatment the usual event appears more

satisfactory, even in severe cases: with convalescence the albumen slowly ceases to be perceived in the urine,* and the disease is thought cured. But presently, on some slight provocation of cold, or irregular diet, the symptom re-appears and gives notice of increasing mischief. This recurrent albuminuria is a characteristic and alarming sign.

There are two practical points intimately connected with this part of my subject: first, that the portions of a kidney left uninjured by the acuter attack are, *ipso facto*, maintained in circumstances of abnormal excitement, and thus predisposed to undergo subsequent change; in other words, that the diseased condition tends to spontaneous increase: and, secondly, that renewed exposure to causes of the original disease,—to intemperance, cold, and the like, will enable these influences to tell with a fearful and accumulative advantage on kidneys already in functional embarrassment. I refrain from expatiating on the clinical details of these cases, which are so frequent in professional practice, and, in their severer forms, so well known. I shall merely remark that any difficulty in their diagnosis may be solved by the microscope. Whenever the disease is advancing, the urine will be found to present in a modified and mitigated degree the same inflammatory products as in the sub-acute attack—the same epithelial cells (more frequently in an immature form), the same occasional (but far less plentiful) presence of the blood corpuscles, and of the fibrinous casts of the urinary tubules.

Thus—with occasional great exacerbations, and with un-failing smaller steps of progress,—the disease may go on, even for years, before continued dropsy and the well-known signs of urinous poisoning give notice of its near termination. But those who are familiar with the course and complications of renal disease will easily believe that a large proportion of

* It is quite certain that the symptom of albuminuria may be absent for some time (probably while the disease is quiescent) even though the kidney be very extensively disorganised.

the patients, whose case I am considering, must die with obscure symptoms not referred to the urinary organs; often from double pneumonia, or from peritonitis, or from encephalic effusion, which is perhaps considered idiopathic, when in fact it has arisen in the non-elimination of urea; and then, on *post-mortem* inspection, the kidneys, often presenting no glaring morbid appearances, are wrongly accounted healthy, and the patient's death is ascribed to a disease only secondary on the renal one.

Now in the *post-mortem* examination of these chronic cases, we may or may not find the kidneys materially contracted and deformed. It happens, to say the least, *very* frequently that the organ has preserved its full size and presents the ordinary colours. Perhaps it may have a cyst or two on its surface. Between such kidneys, and those which are all knobbed and puckered and wrinkled,—there is not the essential difference which first sight would suggest. I shall first detail the changes which are latent in the healthier looking kidney, and subsequently shall consider the anatomy of the contracted specimens, and analyse the circumstances which determine that apparent atrophy of the gland.

In the first instance, then; In commencing the microscopical examination of the cortical substance, we partially find a similar state of tubes to that described in connection with the sub-acute attack—a state, namely, of unequal distention and of blocking up by their own accumulated products. In the cases which have lasted a long time, these products will often be found to have undergone material alterations, from the combined effects of pressure and absorption. The contents of the epithelial cells will have lost much of their natural fine granularity; so that the cells will appear, even when viewed singly, to have acquired a marked increase of solidity and substance. But—more than this; in many parts hardly a trace of tubularity will be found; the tubes have been burst; their contents have been interfused amid the matrix and blood-vessels; and their débris may be found

on opposite sides of a preparation,—here black and bloated, there pale and collapsed.

Betwixt these trophies of disease there is a new manifestation. The interspace is crowded with a profuse development of cysts, apparently foreign to the healthy structure of the part. They are of all sizes; some are visible to the naked eye; some are of the magnitude of normal gland-cells, $\frac{\text{inch}}{1500-1000}$; but the majority are of an intermediate bulk, $\frac{\text{inch}}{300-500}$. Even where smallest, they are distinguished by their sharp outline; and the larger ones are conspicuous by their roundness and transparency, for all above $\frac{\text{inch}}{1000}$ have predominantly fluid contents.

To explain this very remarkable phenomenon I take leave to digress for a moment from the straightforward pursuit of the inflammatory changes. Any one who has made a dozen *post-mortem* examinations must have observed cysts in the kidney; and there can be few pathologists who have not speculated on the origin of these growths. Their connection with chronic obstructive diseases of the kidney being notorious, some observers have supposed them to originate in dilatation of the Malpighian capsules; while others have referred them to distention of the urinary tubules. They exhibit great variety in size; they are seen every day as small as mustard seeds; they have been seen as large as cocoanuts. Thus, they obviously range from a very conspicuous largeness to a size at which the naked eye loses them. On microscopical examination of cysted kidneys, the same uninterrupted gradation of size is seen to repeat itself. The larger vesicles fill the field of the microscope; the smaller ones diminish progressively, so that scores of them may be in the field at the same time.

A section of cysted kidney, carefully examined with a sufficient magnifying power, may show an astonishing number of these minute vesicles; a number quite disproportionate to that of the larger cysts visible to the naked eye; so that sometimes, by a single one of the latter class seen on the surface of the kidney, I have found myself guided to a

disease which is substantially a vesicular transformation of the ultimate structure of the gland. The smallest cysts are simple nucleated cells, of the same size (or rather, within the same limits of size) as the common secretory, or epithelial cells of the gland. From these cells they seem to be distinguished by their very definite outlines, and by their transparent fluid contents: but a step further in microscopical analysis shows that the distinction ceases at this point. They show no signs of a specific origin; no germs can be found for them other than might equally belong to epithelial development; it seems as though from the same germs—according, no doubt, to varying influences—healthy gland-cells might grow, or these fluid-holding cysts.

Fuller investigation of the specimen reveals the following very suggestive fact. The copious formation of cells occupies the place of tubes, holding their relation to the vascular plexus of the gland; and as one gets to the periphery of the portion of gland thus transfigured, one finds the broken extremities of the original tubules,—some empty and collapsed, others obstructed and often dilated with morbid accumulation. In some cases this obstructive material contains a large proportion of fat, or consists of it almost entirely.

In short, in pursuing the minute anatomy of the cysted kidney, we are conducted back to that same structural change which we found in connection with sub-acute nephritis, and demonstrably dependent on inflammatory processes: or sometimes we are led to a change in some respects similar to this, associated with what is known as the mottled condition of the kidney.

The pathology of cysted kidney may accordingly be traced in either of two directions; from its first causation, or from its extreme phenomena. Following the latter course, we have ascended to a period in the history of cysts, in which they lie with numberless gland-germs amid the remnants of broken tubules. The unbroken tubules around show no growth of such cysts in their interior; many are distended, it is true, but not with cysts; their distention is of a kind that we have

already investigated,—inflammatory, or perhaps fatty. From the smallness attained by the cysts, it seems quite obvious to me, that they cannot commence in any transformation of the tubes themselves, or of the Malpighian capsules. Accordingly, I find the same theory suggested by this method of inquiry, as when the morbid change had been traced, descensively, from its causes: viz., that certain diseases of the kidney (whereof sub-acute inflammation is by far the most frequent) tend to produce a blocking of the tubes; that this obstruction, directly or indirectly, produces rupture of the limitary membrane; and that then, what should have been the intra-tubular cell-growth continues with certain modifications as a parenchytic development.*

The only theoretical alternative lies in the supposition that the cysts may have grown in the tubes (independently of their natural contents), and may, themselves, by their development, have caused the destruction of the limitary membrane. But to this theory there are, as seems to me, two insuperable objections; (1,) the absolute invisibility of any such growth within the tubes, and (2,) the still more convincing fact, that the cyst formation does not occur idiopathically, but only in conjunction with diseases which are in themselves fully adequate to account for the blocking and bursting of the tubes.

During the growth of the cysts, they frequently exhibit an endogenous formation of cells which line them as an epithelium: and in this condition (which commonly does not

* I take this opportunity of remarking that true cystic degeneration does not seem ever to result directly from obstruction in the pelvis of the kidney, or in the urethra, or ureters. I have often seen all the receptacular and excretory parts of the apparatus largely distended, without any material injury to the cortical part of the gland: and even in those cases where long-continued distention of the pelvis and calyces has caused expansion and atrophy of the cortex, the formation of interstitial cysts has not presented itself to me. Rayer has judiciously distinguished these latter cysts from the mere dilatation which attends retention of urine in the excretory parts of the kidney; giving to the latter disease the name of *hydrops nephrosis*.

continue if they become very large) they present a curious similarity to the vesicles of the thyroid gland, and are, no doubt, like these, organs of secretion.

If I am right in my statement of facts, and if my theory of the cyst-growth is sound, then the early stages of the process are certainly points of great interest. For no one accustomed to the interpretation of nature can doubt the reparative tendency of these acts. The effused gland-germs are the last phenomena of the original disease, and the first of the attempted compensation. The transparent nucleated cysts, with their clear sharp outlines, are not mere dropsical epithelia ; but are organised for secretion into their own cavities, so as at least to withdraw from the blood, if they cannot eliminate from the body, the materials which fill them.* Nor will it be without interest to the physiologist to remark, as the proximate cause of this peculiar development of gland-germs in the kidney, a modification of structure so slight as the rupture of that limitary membrane, which had heretofore invaginated the cells and had qualified their access of nourishment.

Returning now to the traces of inflammation in an uncontracted kidney, we have yet to ascertain the condition of its blood-vessels. Numbers of the Malpighian bodies are extinct for all purposes of secretion ; their vessels obliterated, their capsules wrinkled round them ; they are dwindle, opaque, and bloodless. Sometimes the contraction of the Malpighian bodies is secondary on that rupture of their capillaries which Mr. Bowman has indicated as the source of intra-tubular haemorrhage ; which rupture, of course, may have arisen either in an augmented impulse of the arterial stream which fills them, or in an impeded circulation through the venous plexus into which they discharge themselves. But rupture of the capillaries is not the only cause of atrophy, to which these bodies are liable in the disease under consideration. The vascular tufts may be exposed to injurious pressure from

* I may take this opportunity of mentioning that on two occasions I have found the Xanthic Oxide forming a considerable proportion of the contents of a cyst.

materials accumulated in their capsules. Thus, I have seen them flattened into a fourth of their natural compass, while the remaining larger portion of the capsule (probably continuous with an obstructed tubule) has been distended with a colourless and transparent fluid.

Such is the minute anatomy of a kidney which, having suffered from sub-acute inflammation, has undergone in consequence no noticeable alteration of volume, although having in its interior a very considerable new development. Now—if that new development could be suddenly subtracted from the volume of the gland, if the cysts could be withdrawn from the parenchyma, what a collapse there would be! how the meshes of matrix would drop together round the atrophied Malpighian tufts, and round the empty tubules of the cortical substance! The kidney would at once be reduced to half or a third of its previous dimensions. The condition thus suggested to the fancy is not without reality; it is one with which every member of the Society is well acquainted, as the extreme stage of renal degeneration,—the stage in which nearly all the efficient elements of the organ have vanished, and have left no new development in their stead.

If the pathology of the uncontracted kidney be rightly understood, that of the contracted specimen will follow it naturally. It seems to me that, in the mere destruction and absorption of tissues, there is abundant explanation of the shrunken dimensions of a kidney which has passed through inflammatory changes. The tubes have burst, and a great portion of their contents has been removed by absorption; the Malpighian bodies have dwindled to a few: what then remains to make bulk? In the uncontracted specimen a false appearance of size is maintained by the adventitious cyst-growth, which I have described as filling the interstices of the organ. But the cysts are so much over and above the real kidney-structure; and if that succulent surplus could be removed, the result, as I have suggested, would be the falling-together of wasted textures into a comparatively small compass. The cause of shrinking in the gland is the gradual absorption of spoiled material. This cause operates equally

in all chronic cases, and its effects are to be traced in the uncontracted, as in the contracted specimen. The main difference between these two lies in the *more or less* of interstitial cyst-development; the most dwindle are those in which least of the new growth has arisen or has survived.

I see no reason for believing that the interstitial effusion of lymph effects much towards the final contraction of the kidney. There are not wanting, I know, some pathologists who will assert it to be the great agent in the change; and who conceive they have seen the whole process of fibre-formation according to the most approved foreign cell-theories. But I suspect that the observers of new fibre will often have confounded cause and effect. Coincidently with atrophy of the kidney there occurs a contraction of the reticular matrix; but *that* contraction is probably consequent on a prior absorption of the intervening tissue. The meshes of the matrix come nearer together, and in a given space there is an excess of fibrous tissue, only because the material is withdrawn which originally expanded that matrix through three times the space it now occupies.

I am by no means prepared to affirm that interstitial effusion of lymph and its subsequent organization may not *possibly* occur in the kidney; or that, if it occur, it may not *in any degree* conduce to the resulting atrophy. Indeed, I have seen what I have thought to be interstitial new matter assuming a certain fibrous arrangement; but this has been very rarely, perhaps somewhat doubtfully; and it has always shown itself in a degree far subordinate to the other causes of contraction. Destruction of tubes and Malpighian bodies, with more or less extensive vascular obliteration, is *always* to be observed in specimens of contracted kidney; and I have never seen a degree of contraction in the gland which these changes might not sufficiently explain. I therefore find no reason for assuming the superfluous operancy of an undemonstrated agent.

In respect of kidneys contracted after the inflammatory process, a very interesting question occurs to me;—Have they passed through an uncontracted cystogenous stage?

and, if so, what has become of so many cysts? I am unwilling to speak very positively on this point, but I suspect that the following observations may explain in some degree the relations of the two forms.

In the uncontracted cystogenous kidney, the disease has been of rapid process; for it has usually invaded the greater portion of both glands, and its course has been short in proportion to its intensity. These are the cases in which the phenomena of the disease are best studied: cases in which an infinite number of cysts occur, ranging upwards from epithelium-size to a magnitude visible to the naked eye, and seldom much larger.

If, on the contrary, the original inflammatory attack and the cystic degeneration have been restricted to smaller portions of the gland, and if (as no doubt very often happens in connection with gout or rheumatism) various portions of the gland have been successively invaded, a different result occurs. The vesicles, if placed advantageously near the surface of the organ, may undergo an ulterior and almost indefinite growth, presenting us with the well-known phenomenon of large renal cysts. But, if placed in the interior of the cortical substance, I am disposed to believe that they must in some measure promote its ultimate demolition. For, their pressure on the capillary plexus which surrounds them and which was originally not adapted for such an arrangement, must, of necessity, tend to impede and ultimately to obliterate it; which vascular obliteration would of course entail their own subsequent atrophy and disappearance. I believe that in contracted kidneys something of this sort must have occurred, and that the cyst-growth, except very partially and under very peculiar circumstances, cannot advance beyond the low degree of development witnessed in the uncontracted specimens. This belief is partly founded on, and would partly explain, the fact, that there is a certain inverse proportion* observed be-

* This fact is not to be explained by the supposition of confluence of several smaller cysts to make a larger one; a method of development which I have never noticed, or even seen satisfactory traces of, and which—if it ever occur, must be of the greatest rarity.

tween the *size* of the cysts and their *number*: as if the same length of time which suffices to give bulk and extreme development to a portion of the crop, determines also the abortion of the rest. I may observe too, in further illustration of the same point, that the contracted kidneys—although they present in their interior an infinitely scantier formation of cysts than the uncontracted ones—do yet, even when most atrophied, rarely, if ever, fail to afford some traces of the cystogenetic process.

Accordingly I am inclined to view this process as inseparable from any such sub-acute inflammation of the gland as may terminate in its contraction. But of cysts thus generated few can attain maturity. They are girt with tissues that impede their free expansion, and are fed by a vascular plexus ill-suited to their nourishment. Their growth soon arrests itself by compressing the blood-vessels that supply it; and thus, while in the slower cases, a few may attain considerable size, the numerical majority will have participated in the general atrophy of the gland.

Up to the present point I have studiously avoided introducing the ambiguous and controversial name of "Bright's disease." And now it will probably be asked, what relation to Bright's disease is borne by the malady I have treated of. Is it the same thing under another name? This question can be answered in a word, only when it shall have been settled what Bright's disease really is. The history of the complaint, or complaints, included under that title was perhaps originally systematised with too much haste. Starting from dropsy with albuminuria, and noticing that two chief forms of morbid appearance corresponded to that symptom (one, namely, where the kidney was large and mottled,—the other where it was contracted and knobbed, or irregularly granular), pathologists have considered these two forms as representing the extreme stages of one and the same disease.

I must venture to express a doubt as to the justice of this generalisation. After investigating both classes extensively, I am convinced that the *mottled* and the *contracted* kidney do,

in almost every instance, belong to different morbid actions, not to different stages of the same. This opinion I shall very briefly defend.

Last session, in one of the soundest and most ingenious papers ever read before this Society, Dr. Johnson contended that Bright's disease is a fatty degeneration of the kidney. He started from *the mottled kidney*, taking it as, *κατ' ξοχὴν*, Bright's disease. Far more fully and conclusively than had hitherto been done, he illustrated the pathological affinities of that disease, and demonstrated its essential connection with the scrofulous diathesis. This view was corroborated by some experiments of my own (which Dr. Johnson did me the honour to quote), in which I had artificially produced a fatty degeneration of the kidney by measures adapted to develop the strumous cachexia. I will not absolutely affirm that mottled kidneys may never undergo atrophy. In certain very few cases where the scrofulous deposit has been made in consecutive small instalments, slowly disorganising the gland—in such, *perhaps*, contraction may have ensued in the portions first and separately invaded. But I am persuaded that this occurrence must be quite exceptional. Under ordinary circumstances the fatty deposit occurs with uniformity throughout the gland, the function of which is so totally disturbed, that death anticipates the leisurely process of its contraction. The mottled kidney, in an infinitely *large* proportion of cases, remains *large* and mottled to the end.

On the other hand, if we start from what has been considered the final stage of Bright's disease, *the contracted and atrophied kidney*, we mount, step by step, not to fatty degeneration of the gland (or, if ever, with peculiar infrequency), but, through the series of changes enumerated in the present paper, to epithelial engorgement, to sub-acute inflammation: we mount to a new circle of pathological affinities, not to the strumous degenerations—not to phthisis and its congeners, but to the irritative blood-diseases, to fever, gout, rheumatism, and the like.

It seems almost indispensable for correct nomenclature

that the name of "Bright's disease" should be discontinued; for I do not see how it can be restricted to either of the two diseases which modern pathologists recognise as included by it. *Stant nominis umbrā.* One class of persons may persist in giving the name to contracted kidneys which never have been mottled; others to mottled kidneys never about to be contracted. Both parties, with equal right or equal wrong, may appeal to the admirable pages of the "Medical Reports," and find their ambiguity apparently justified. Each of the two diseases is "Bright's disease;" for both are described with equal originality and truthfulness by the respected physician whose name has been identified with them.

I would suggest that, for the one class of cases which I believe to be far most numerous—the class, namely, treated of in the present paper—it will be convenient to employ the familiar and appropriate name of INFLAMMATION. I have chosen to call it *sub-acute*, rather than *chronic*, because its peculiarities have more to do with low intensity than with long duration. Cold and intemperance, the various fever-poisons, the irritation of gout, of rheumatism, of the oxalates,—these are its habitual causes. Its tendency is to the obliteration of tubes, to the development of parenchymatic cysts, and finally to the contraction of the kidney.

Secondly,—There will be the degeneration which Dr. Johnson so successfully illustrated last session, commencing, as he demonstrated, in a fatty engorgement of the epithelium, and therein distinguished from the purely inflammatory affections. In its progress this disease has invariably appeared to me to have the sub-acute inflammation grafted on it; but *its origin is sui-generis*, is non-inflammatory; and it would be convenient, I think, if it were separated from the other under the name of SCROFULOUS DEGENERATION. This disease, as I have stated, leads in very few cases to contraction of the gland: at least, such is my own experience, and I have obtained from others some very strong evidence confirmatory of my opinion.

I may add that I have often seen the interstitial cyst-development in connection with the scrofulous degeneration, but not so frequently or regularly as in the sub-acute inflammation of the kidney. I am unable at present to decide whether the cyst formation belongs in these cases to the fatty disease, as of *its* creation; or whether it merely arises when the inflammatory process has been superinduced, and depends for its birth (just as all the other fatal and irreparable changes in the gland depend for *their* immediate causation) rather on that secondary influence than on the original accumulation of fat.

I have now little further to add. With respect to the symptoms of sub-acute inflammation of the kidney, I will make one observation in addition to those already embodied in my paper. The descent of epithelium and its germs with the urine; the presence of albumen there, and sometimes of blood; the little casts of the tubules—sometimes wrought of fibrin, sometimes of compressed epithelium;—these signs belong equally to the sub-acute inflammation and to the scrofulous disease. They are signals simply of renal irritation, whether from one cause or the other, and I suspect they only attend the scrofulous disease at that stage of its progress in which sub-acute inflammatory action is superadded to the primary fatty degeneration. Dr. Johnson's accurate observation has enabled us, under most circumstances, to diagnose the two classes from each other; for, in the scrofulous disease there will be always seen, as he describes, more or less oil entangled in the fibrinous casts, or gorging the cells which descend in the urine; a phenomenon which does not belong to the pure sub-acute inflammation.

In regard of treatment; I have no new drug to recommend for the removal of cysts or the restoration of tubules. Here, as with so many other maladies, vigilance with respect to the first premonitions of the disease, and an exacter knowledge of its pathology, will enable every intelligent practitioner to apply old remedies with improved success.

From the moment in which albumen exists in the urine, I believe the exhibition of diuretics to be perilous and unwise in the extreme. From that moment everything must be done to spare and economise the injured organ.

Weight in the loins, with increased disturbance of the secretion, will often suggest the expediency of abstracting blood, locally, by cupping. Where the necessity of this measure is indicated, and where it can be borne, it never fails to give relief. In cases which seem unable to bear abstraction of blood, dry cupping is often attended with advantage.

In no remedy am I disposed to place so much confidence as in the sweating-bath; whether by dry air or watery vapour. I believe that its habitual use tends, more than any system of treatment with which I am acquainted, to counteract the chronic causes of the disease, to retard its progress and palliate its effects.

That cold must be most carefully guarded against; that every gastric source of renal irritation must be studiously obviated by measured diet and appropriate medicine; that dropsy and other secondary affections will require their own specific treatment;—all this is so obvious, that it would be worse than superfluous if I should detain the Society by dwelling on it. The treatment is a plain, common-sense inference from the pathology.

To conclude; a disease is practically important, in proportion as it is frequent or fatal. That on which I have so long detained the Society has both these claims: it is peculiarly frequent, and, in its confirmed form, of necessity shortens life.

When I remind the Society of its causation; how exposure to cold, how gout, rheumatism, scarlatina, cause it; how, too, not only scarlatina, but other blood-poisons almost as frequently;—it will be obvious that few can arrogate to themselves an entire immunity from the causes of the disease. That these causes need act with no great intensity in order to their destructive result, will be evident from the observations

I have made on epithelial engorgement of the kidney as a means of its frequent disorganization.

Sub-acute nephritis has appeared to me to exist quite as frequently as pulmonary consumption; and I shall not, I think, exaggerate in saying, that in two-thirds of its cases it is latent: its symptoms are overlooked during life, partly from their unobtrusiveness, partly because they are often merged in those of some striking constitutional disorder; while in like manner its organic traces in the dead body have been neglected, as rarely impressing on the kidney those grosser changes of aspect which would arrest the casual observer. I repeat that it has happened to me again and again, after *post-mortem* examinations, to find, in kidneys roughly estimated as healthy, the most thorough abolition of natural structure.

And I shall be satisfied with the result of this paper, if the Society hereafter be convinced, that a disease of immense frequency and of the greatest seriousness has hitherto, in a majority of its circumstances, escaped the observation it deserves.

For assistance in obtaining materials for the present paper, I am much indebted to my Hospital-colleagues; to Dr. Liddell, Inspector of Hospitals; to Dr. Cohen, of St. Thomas's; and to Messrs. Nunn and Brinton, my colleagues at King's College. I am anxious peculiarly to express my obligation to Mr. Prescott Hewett, not only for his remarkable kindness in furnishing me with many important illustrations for my paper, but likewise for the access allowed me to that excellent collection of reports which he has organised at St. George's Hospital, and which I believe to be unequalled in this country.

Note.—The doubt I have ventured to express as to the so-called *stages of Bright's disease*, and especially as to the

alleged *contraction of the mottled kidney*, first suggested itself to me in the form of the following argument.

The mottled, or fatty, kidney is essentially scrofulous: it is constantly found as a complication of phthisis. If its tendency be to produce contraction of the gland, or rather if it be but the first stage of a disease habitually advancing to that result; then, unquestionably, contracted kidneys ought to be found in the necropsies of phthisical patients. Are they so found?

In referring to statistics on this subject, I may remark that there is less room for fallacy than in many such numerical quotations. The diseased condition is very obvious. Mottled kidneys may mistakenly be written healthy, and the observer who has erred in respect of one specimen will probably err again with many. But no moderately careful person can possibly call a contracted kidney a healthy one.

Accordingly, I expect to learn, at least approximately, the disposition of the scrofulous degeneration to induce contraction of the gland, when I ask the following question:—In how many instances of fatal phthisis has a contracted kidney been seen?

1. Monsieur Louis refers to 214 examinations of the kidney in phthisis. In no one of them does he report any diminution of size to have been noticed.

2. In the invaluable Museum Book of St. George's Hospital, among 116 cases, carefully and circumstantially registered, I find only two that require notice; one, where in a woman of 42, dead with vomicae in the lungs, both kidneys were found "small, particularly the right one which had a granular surface;" and another of a man (age not mentioned) dead of phthisis, whose kidneys were "much reduced and granular, with a large cyst in the left one." Unfortunately there were no particulars to be obtained of these patients' histories, nor had any microscopical examination of the specimens been made. I mention these two, though they prove nothing one way or the other, as being the only two out of 116 phthisical cases to which the slightest suspicion of im-

portance could attach. I may add, too, that Mr. Hewett, who assisted me with these references, and to whose opinion on such a subject the utmost deference is due, tells me that he has long entertained doubts as to the contraction of kidneys affected with scrofulous degeneration; that he believes (as I do) that the *mottled* and *contracted* kidneys are of different pathological families.

EXPLANATION OF PLATE V.

This plate illustrates Mr. Simon's paper on Sub-acute Inflammation of the Kidney.

1, 2, 3, 4, 5, 6.—Different appearances of the cyst formation.
1, 2, 8.—Unnatural distention and plugging of the tubules.
7.—The vascular tuft of a Malpighian body squeezed into small compass by fluid pressure from within the tube.
1 and 3 are seen with an eighth object-glass; and the smaller cysts, here represented, are not larger than ordinary epithelial cells; the others are seen with a fourth-inch object-glass.



3



4



6



ON THE
INFLAMMATORY DISEASES OF THE
KIDNEY.

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IN a paper published in the last volume of the Society's Transactions I gave some account of fatty degeneration of the kidney, and declared my intention to make the inflammatory diseases the subject of a separate communication. On the present occasion I purpose to bring before the Society the result of some observations on this very interesting and important subject.

The inflammatory diseases of the kidney may be arranged in two distinct classes: — the first class including those diseases which are the result of some local cause—such as retention of the urine in consequence of stricture, the mechanical irritation of a stone, or a blow on the loins: while in the second class are included those diseases which are the result of a constitutional cause, or, to speak more precisely, of some abnormal condition of the blood. The latter class of diseases is by far the most important, and it is this class only which will be treated of in the present communication.

In the paper before alluded to, when referring to the condition of the kidney which occurs as a consequence of searlatina, I stated that "it is in fact an inflammation of the kid-

ney, excited like the inflammation of the skin which constitutes the eruption of scarlatina, by the passage through the part of the peculiar fever poison; and, as the inflammation of the skin terminates in an excessive development of epidermis and a desquamation of the surface, so the inflammation of the kidney excites an increased development of the epithelium which lines the urinary tubules: this material partly accumulates in and chokes up the tubes, while part of it becomes washed out with the urine, and may be detected in large quantities in that fluid by the aid of the microscope."

To the account then given, which I believe to be essentially correct, subsequent observations enable me to make some important additions. The appearance of the kidney in these cases is admirably represented in a drawing which was made for me by Dr. Westmacott.* The kidney was taken from a girl who died in a few days after symptoms of renal disease had manifested themselves. The gland is enlarged, apparently by the deposit of a white material in the cortical substance: the vessels in the cortical portion, where they are not compressed by this new material, are injected and of a bright red hue; the pyramidal portions are of a dark red colour, in consequence of the large veins which occupy these portions of the gland being distended with blood. The appearance of the entire organ is quite that of a part in a state of acute inflammation.

When the kidney has been in a softened condition before the occurrence of the inflammatory disease, as often happens in elderly persons, the lobules on the surface appear larger and coarser than natural; the veins, being less compressed than when the natural texture of the kidney is firmer and more unyielding, are much distended with blood, so that the entire organ is of a dark slate colour. (Dr. Bright's fifth plate represents a kidney in this condition.)

On a microscopical examination, the convoluted tubes are

* The drawings referred to here and elsewhere in this communication were exhibited at the meeting, and are now in the possession of the author.

seen filled in different degrees with nucleated cells, differing in no essential character from those which line the tubes of the healthy gland. The chief difference between these cells which are the product of inflammation, and those which exist in health, consists in the former being generally of smaller size and more opaque and dense in their texture. It is very interesting and important to observe that, while the convoluted tubes are rendered opaque by this accumulation of cells in their interior, the Malpighian bodies are transparent and apparently quite healthy. (See Plate VI. fig. 1.) The straight tubes which form the pyramids also contain an increased number of cells, but there is reason to believe that these cells are not formed in these portions of the tubes, but that they are lodged there in their passage from the convoluted through the straight tubes, the latter being merely ducts leading into the pelvis of the kidney. Some of the tubes contain blood, which has doubtless escaped from the gorged Malpighian vessels lying within the dilated extremities of the tubes. There is no deposit outside the tubes. The essential changes in the kidney are an increased fulness of the blood-vessels, and an abundant development of epithelial cells, differing slightly in general appearance, size, and consistence, from the normal renal cells; this increased cell-development occurring in those portions of the urinary tubules, the office of which, as Mr. Bowman has suggested, is to excrete the peculiar saline constituents of the urine, while the Malpighian bodies, whose office is the separation of the water, are unaffected.*

* I may here mention an observation which is interesting in connection with the pathology of nephritis, and which affords additional evidence as to the accuracy of Mr. Bowman's theory. Within the last few months I have examined the kidneys of two persons who died jaundiced, and in whose urine there had been a large quantity of bile. Many of the urinary tubes were stained of a deep yellow colour by the bile in their epithelial cells. This yellow colour ceased abruptly at the neck of the Malpighian bodies, and in no instance did I observe it in the Malpighian bodies themselves.

The condition of the urine in these cases is clearly indicative of the changes occurring in the kidney. After the urine has been allowed to stand for a short time a sediment forms, and on placing a portion of this under the microscope, there may be seen blood-corpuscles, with epithelial cells in great numbers, partly free and partly entangled in cylindrical fibrinous casts of the urinary tubes;* and very commonly numerous crystals of lithic acid are present. (See Plate VI. fig. 7.) As the disease subsides, which under proper treatment it usually does in a few days, the blood, fibrinous casts and epithelial cells diminish in quantity, and finally disappear; but traces of the casts and cells are still visible, some days after the urine has ceased to coagulate on the application of heat or nitric acid.

The casts and cells, which appear in the urine when the disease is subsiding, are such as have remained some time in the urinary tubes before they have become washed out by the current of fluid poured into the tubes from the Malpighian bodies: many of the cells entangled in these casts have, consequently, become disintegrated and broken up into amorphous granular masses, thus presenting appearances which I shall presently show are characteristic of the casts occurring in cases of chronic nephritis. Such is the morbid anatomy of the kidney, and such are the characters of the urine occurring as a consequence of searlatina.

The present state of physiological science enables us to give a sufficiently satisfactory explanation of these phenomena. It is now a well-known and well-established fact, that all secretion is performed by the agency of nucleated cells,—cells which, in process of growth and development, abstract certain materials from the blood, which materials constitute the secretions of the various glands. While there is a certain general *resemblance* between the cells of all true glands, there are also certain well-marked *differences* between the cells of different glands. For example, the hepatic and

* These fibrinous casts of the tubes were first observed in the urine by Dr. J. Franz Simon.

the renal cells have certain characters common to both, as well as certain distinctive characters which would enable a practised observer at once to distinguish one from the other. There is thus a mutual adaptation between particular secretions and particular secreting cells. The hepatic cell is adapted, in a manner beyond our comprehension, for the separation of biliary matter, and the renal cells for the separation of urea and the other solid constituents of the urine: and again, the biliary matter and urea, &c., are the materials adapted for promoting the growth, development, and final disappearance and blending with the secretions, of such cells as the hepatic and renal respectively.

The increase in the number of slightly altered epithelial cells in cases of renal disease consequent on scarlatina, is a certain indication that materials not naturally forming a part of the renal secretion are being excreted by the kidney. These materials are doubtless combined with the cells, and their separation from the blood is effected by the growth and development of the cells. The increased quantity of blood in the kidney, and the increased cell-formation, are mutually connected; in like manner, the ascent of the sap and the expansion and development of the leaf-buds are mutually connected and inter-dependent.

The presence of albumen and blood in the urine is obviously a result of the turgid condition and the active congestion of the entire vascular system of the gland. As this condition of the vessels in any of the tissues of the body commonly leads to extravasation of serum or even of all the constituents of the blood into the surrounding parts, so the structure of the kidney, and particularly that of the Malpighian bodies, is obviously such as renders the admixture of blood or serum with the urine a necessary consequence of the before-mentioned condition of the vascular system.

The imperfect elimination of the urinary constituents, their accumulation in the blood and the consequent deterioration of this fluid, are the obvious and necessary consequences of the choking up of the tubes of the kidney, and of

the obstacle thereby offered to the efficient performance of their secretory function. So great may be the interference with the excretory office of the kidney, that the disease may prove fatal within a few days, by the occurrence of coma or of inflammation of one or more internal organs. Under judicious treatment, however, the majority of these cases recover, the noxious matters are effectually eliminated, the vascular congestion and the desquamation of the urinary tubules simultaneously diminish, the cells which were thrown into the tubes are gradually washed out, and the kidney is completely restored to its original, healthy condition.

To the form of renal disease here described as occurring in connection with scarlatina I propose to give the name of "*acute desquamative nephritis*."

The question next arises,—Does this condition of kidney occur in any cases unconnected with scarlatina? A careful observation of several cases during life, and a comparison of the appearances presented by the urine with the condition of the kidney after death, enable me to state with confidence, that a form of renal disease (differing in no essential respect, either as regards symptoms or anatomical appearances, from the "acute desquamative nephritis" which occurs in connection with scarlatina, is very commonly met with unconnected with scarlatina. A detail of the cases upon which my conclusions are founded would render this communication inconveniently long; I shall therefore content myself with a brief statement of the general results.

In children, by far the most frequent cause of acute desquamative nephritis is scarlatina; in adults, it occurs less frequently as a consequence of scarlatina, but much more commonly as a consequence of other conditions which are known to exert a powerful influence on the general nutrition of the body, as well as on the process of assimilation and of functional integrity in particular tissues and organs.

The first case in which I detected acute desquamative nephritis, unconnected with scarlatina, was in a girl named Eliza Smith, *atæt.* 21, a patient of Dr. Budd's, in King's

College Hospital. She had suffered much from secondary syphilis, and, at the time of her admission on the 26th Nov. 1845, was very weak and emaciated, had cough with mucous expectoration and night sweats, and had very painful nodes on the shin bones. She soon began to improve under the use of Iodide of Potassium and a nutritious diet. In the early part of December, symptoms of renal disease were first observed ; she had frequent vomiting, and on the 16th Dec. the urine was found to be scanty and very albuminous. The day before this, she was seized with pleurisy on the left side. These symptoms continued until the 11th January, when she died. The kidneys presented all the characters of "acute desquamative nephritis," and there was a sero-purulent liquid in the left pleura.

In another case the disease appeared to arise from mental anxiety, combined with insufficient food ; and in several others it could be traced to a want of sufficient food, with the addition, in some cases, of bodily fatigue and exposure to wet or cold.

The *Medical Times*, of May 22nd, 1847, contains the report of a very interesting case, evidently of acute nephritis supervening upon the suppression of an extensive eruption of impetiginous eczema. The case occurred in the Hospital Saint Louis, and is thus reported :—" R—, ætat. 27, a porter, was admitted into the hospital on the 10th March last. Some months previously, under the influence of circumstances which remained unknown, a scaly eruption showed itself on the body and limbs : warm-baths were exhibited ; the scales fell, and an eruption of impetiginous eczema appeared on the thighs, scrotum, and arms. The eruption was rapidly yielding to a mild antiphlogistic treatment, when, on the 12th April, œdema of the face, intense pain in the lumbar region, and suppression of urine, were observed, together with a considerable degree of dyspnoea and fibrile excitement. In the peritoneal cavity a certain amount of effusion was also detected. The urine, which at first was altogether suppressed, was afterwards secreted, though very scantily, and was found to contain a large quantity of albumen. The

respiration became more and more difficult, although auscultation of the chest and heart betrayed the presence of no physical change; and after three days the patient died. The treatment consisted in venesection on the first day, and afterwards of abundant drinks with nitre and syrup of squills, and of stimulating frictions with croton oil on the region of the loins. Tartar emetic was also exhibited without procuring any relief. On dissection, a small quantity of transparent serum was found in the peritoneum: the kidneys, otherwise healthy in their texture, were slightly injected on their surface: the lungs and heart presented no morbid change, and no alterations were detected in the brain or spinal cord."

I shall have occasion to refer again to the connection between cutaneous and renal disease.

The predisposing cause of the disease in all these cases is the presence, in the system, of abnormal and irritating products, the result of mal-assimilation. The kidney suffers in the manner already described, during the process of excreting these abnormal products, which are often determined to the kidney suddenly and in great quantity by exposure to cold and the consequent impairment of the cutaneous functions; so that it commonly happens that the immediate exciting cause of an attack of acute nephritis, with dropsy and albuminous urine, is exposure to wet or cold: and this is true of the cases occurring in connection with scarlatina, as well as of those unconnected with this disease. It is highly probable that exposure to wet and cold would never produce that form of disease under consideration, if the person so exposed had been previously healthy and well nourished. It is the *quality* of the blood sent to the kidney, and not the *quantity* of this material, which produces the disease in question.*

The next form of inflammatory disease, to which I would

* I am desirous of acknowledging the assistance which I derived from Mr. Simon in making my earliest examinations of the kidney in cases of dropsy after scarlatina. Some later as well as more important and satisfactory observations on the same subject were made conjointly by Dr. Todd and myself. A short account of these observations was given by Dr. Todd

direct attention, is one of great interest and importance. Two drawings by Dr. Westmacott represent the disease in two different stages: one represents a kidney in the earlier stage; the other shows a more advanced stage of the same disease. The kidney is never much enlarged; in the earlier stage the size of the organ is natural, and the structure of the cortical portion appears confused, as if from the admixture of some abnormal product; there is also some increase of vascularity. As the disease advances, the cortical portion gradually wastes, the entire organ becomes contracted, firm and granular, the pyramidal bodies remaining comparatively unaffected even in the most advanced stages: simultaneously with the diminution in size of the kidney there is a decrease of vascularity. These changes occur very gradually: the disease is essentially chronic, having a duration in most cases of many months, and in some even of several years. It is almost confined to persons who are in the habit of partaking freely of fermented liquors; it is very commonly seen in those who have suffered from gout, and is not uncommon in those who, having indulged freely in the use of fermented liquors, have yet never had an attack of gout. It is sometimes, but I believe rarely, met with in those whose mode of life has been strictly temperate and abstemious. The symptoms usually attending the disease are the following:—*Dropsy*, which

in a clinical lecture published in the *Medical Gazette* for December 1845. In the same lecture Dr. Todd remarks, “the anatomy of the kidneys in inflammatory dropsy (unconnected with scarlatina) has yet to be investigated. I believe I can furnish a contribution to the subject; but as my examination was made a long time ago, I do not desire to attach great importance to it.” Dr. Todd then reports the case of a fine-looking young man, 25 years of age, who, after exposure to cold, had an attack of dropsy with albuminous urine. At the end of a month the patient died under an attack of pleurisy and peritonitis. Dr. Todd thus describes the microscopic appearances of the kidney: “The tubes were enlarged and filled by an abundant epithelial formation, which seemed to distend them; on making a transverse section, the dilated state of the tubes was more conspicuous, and the epithelium could be pressed out of them, leaving only their dilated walls. Several Malpighian bodies were enlarged.”

commonly is not excessive, often coming on only in the most advanced stages, and sometimes being entirely absent throughout the entire progress of the disease. The urine is commonly albuminous; it seldom, however, contains a very large quantity of albumen, and sometimes there is no coagulation on the addition of heat or nitric acid. The urine is sometimes high-coloured and scanty, but in most cases it is rather abundant, pale, and of low specific gravity—from 1005 to 1010. In some instances the quantity of urine is much greater than in health, and this increased quantity of urine is secreted by kidneys which are found after death to be contracted to one third of their original bulk. In urine of such low specific gravity there is of course a deficiency of the solid constituents, while the blood, which is much changed and impoverished, contains an excess of these materials.

On a microscopical examination of the kidney, the nature of the above-mentioned changes is very clearly revealed, and at the same time the attending symptoms are satisfactorily explained. My account of these phenomena will be rendered more intelligible if I give the facts and their explanation at the same time.

On placing thin sections of the kidney under the microscope, some of the tubes are seen to be in precisely the same condition as in a case of acute desquamative nephritis; they are filled and rendered opaque by an accumulation within them of nucleated cells, differing in no essential respect from the normal epithelium of the kidney; this increase in the number, and this slight alteration in the character, of the epithelial cells, are the result of the elimination by the kidney of mal-assimilated products, which are being continually developed in these gouty and intemperate subjects, and which are not normal constituents of the renal secretion.

There must evidently be a certain limit to the number of cells which can be formed in any one of the urinary tubes, for although some of the cells escape with the liquid part of the secretion, and so may be seen in the urine, as in a case of acute desquamative nephritis, yet in many of the tubes the

cells become so closely packed that the further formation of cells is impossible, and the process of cell-development and, consequently, of secretion within that tube, are arrested. The cells, thus formed and filling up the tube, gradually decay and become more or less disintegrated. While these changes are going on in the convoluted portions of the tubes, the Malpighian bodies remain quite healthy, the Malpighian capsules for the most part transparent, and the vessels in their interior are perfect. (See Plate VI. fig. 1.) From these vessels, water, with some albumen and coagulable matter, is continually being poured into the tubes ; and, as a consequence of this, the disintegrated epithelial cells are washed out by the current of liquid flowing through the tubes, so that, on examining the sedimentary portion of the urine, we find in it cylindrical moulds of the urinary tubes, composed of epithelium in different degrees of disintegration and rendered coherent by the fibrinous matter which coagulates amongst its particles. The appearance of these casts, which are quite characteristic of this form of "chronic desquamative nephritis," is represented in figs. 10, 11 and 12.

There is reason to believe that when the process of cell-development and of secretion have once been arrested, by the tube becoming filled with its accumulated contents, it never recovers its lining of normal epithelial cells ; but when the disintegrated epithelium has become washed away from the interior of the tube, the basement membrane may be seen in some cases entirely denuded of epithelium : in other cases a few granular particles of the old decayed epithelium remain (see Plate VI. fig. 2) : and again in other instances, the interior of a tube which has been deprived of its proper glandular epithelium is seen lined by small delicate transparent cells, very similar to those which may sometimes be seen covering the vessels of the Malpighian tuft. (See Plate VI. fig. 3.)

It now becomes interesting to ascertain what further change the tube undergoes, after having lost its normal epithelium. It is quite certain that, as a general rule, the Malpighian bodies remain unaffected, both in structure and in

their office of secreting the watery constituents of the urine, until the whole of the disintegrated epithelium has been washed out of the tubes. Of this there are two proofs; the first is the fact of a very long convoluted tube having its contents completely washed out, and its basement membrane left quite naked: this could happen only as a consequence of a current of liquid passing through the tube, and there is no known source of such a current but the Malpighian vessels: the second proof is still more convincing and satisfactory, and it is this,—that a tube may often be seen entirely denuded of its epithelial lining and continuous with a Malpighian body, in the interior of which the vessels are quite perfect.

Now, a tube of this kind deprived of its lining of normal epithelium has manifestly lost its power of separating from the blood the solid constituents of the urine, while, the Malpighian vessels remaining unaffected, the power of secreting water remains. Further, it appears probable not only that the Malpighian body continues to secrete water, but that the whole length of a convoluted tube thus deprived of its proper epithelium, and either remaining naked or lined by delicate nucleated cells, such as those which cover the Malpighian vessels—that the entire length of such a tube becomes a secretor of water, which it abstracts from the portal plexus of vessels on its exterior. This is rendered probable by the appearance of the tube itself, and the probability is still further increased by the fact of the tubes becoming in some cases dilated into cysts, which usually contain a simple serous fluid without any of the solid constituents of the urine.

It has long been supposed that the simple cysts, which are so commonly seen in connection with some forms of renal disease, are in fact dilatations of the urinary tubes. I am not aware that any satisfactory evidence has been adduced in confirmation of this opinion, but there are some facts and arguments which appear to me abundantly sufficient to prove the accuracy of the notion.

1st. The tubes thus denuded of their epithelium are often seen much dilated. I have repeatedly seen them three or

four times exceeding their normal diameter,—in some cases the dilatation is very sudden, so that the tube assumes a globular form, and appears to bulge in the intervals of the fibro-cellular tissue in which the tubes are packed. (See Plate VI. fig. 5.) In some cases, too, the basement membrane appears thickened in proportion to the dilatation of its cavity. Now, this process of dilatation having once commenced, and the lower end of the tube becoming closed by a deposit in its interior or by pressure from without, there is no reason to suppose that the process may not continue until a cyst as large as a pea or a walnut is formed.*

2ndly. But there are other facts which afford a very interesting and remarkable confirmation of this notion. In a case of simple acute or chronic nephritis, the quantity of oil in the *secreting cells* of the kidney is very small; sometimes indeed none can be detected. But it frequently happens that after a tube has been stripped of its secreting cells in the manner before mentioned, an accumulation of fatty matter occurs in its interior, the denuded basement membrane becomes scattered over with separate oil-globules, and these increase in size until they form masses of fatty matter having much the appearance of adipose tissue; and such a mass is frequently washed out from the tube and may be detected in the urine.† This occasional filling of the tube with fatty matter is very interesting, in connection with the

* The precise cause of obstruction may frequently be seen on examination of the tubes about the bases of the pyramids; the tubes which occupy this position are frequently found to be completely obstructed by the epithelial particles which have been washed into them from above, that is, from portions of tubes nearer the Malpighian bodies.

† The appearance of these clusters of oil-globules is so very different from that presented by the *secreting cells* when distended with oil (see Med.-Chir. Trans., Vol. xxix., Plate I.), that their presence in the urine by no means increases the difficulty in the diagnosis between a fat and an inflamed kidney; on the contrary, the detection in the urine of such a mass as is represented in Plate VI. fig. 8, would of itself justify the inference that the kidney from which it had come was in a state of chronic inflammation rather than of fatty degeneration.

fact that in some cases the cysts, which are supposed to be dilated tubes, are also found filled with the same material. In two cases I have found a cyst as large as a hazel-nut quite full of oil, presenting all the characters of that seen in the tubes which have lost their epithelium in consequence of chronic inflammation.

The evidence, then, of the simple serous cysts being dilated tubes is the following:—1st. The tubes are often seen much dilated and thickened. 2nd. As the inner surface of the tubes has the appearance of being endowed with the power of secreting water, so the cysts usually contain a simple serous fluid. 3rd. As an accumulation of oil occasionally occurs in the tubes, so the cysts are in some instances filled with the same material. 4th. *There is no reason to suppose that these cysts have any other origin.* It appears probable that the Malpighian bodies could not become dilated into cysts, because an accumulation of liquid within the Malpighian capsule would necessarily compress and obliterate the vessels of the Malpighian tuft, and so would cut off the further supply of fluid.*

Another change consequent upon the destruction of the cells which line the urinary tubes is, a diminished supply of blood and a gradual wasting of the tube. I have already shown that there must be a close connection between an increased development of epithelial cells and an increased afflux of blood to the part. This is well seen in a case of acute desquamative nephritis, and, *vice versa*, a more or less complete destruction of the epithelial cells will be attended by a corresponding diminished afflux of blood and a consequent atrophy of the part affected. In every kidney which has been the subject of chronic inflammation, there may be seen tubes contracted in different degrees as a consequence of

* Additional evidence as to the nature of these cysts, and their relation to the normal tissues of the kidney, is afforded by some remarkable preparations of diseased kidney in the Pathological Museum of King's College. See the Author's Catalogue of the Museum, Nos. 871, 872, 873, 874, and 875.

the destruction of their epithelial lining: in some instances the basement membrane becomes folded, and presents an appearance not very unlike white fibrous tissue. As a consequence of this wasting of successive sets of tubes there is a gradual diminution in the bulk of the cortical portion of the kidney, until at length the entire organ becomes small, contracted and granular. When a thin section of a kidney thus atrophied is placed under the microscope, there may be seen an abundance of fibrous tissue, and this has often been described as new fibrous tissue developed during the progress of the disease, whereas it is in reality nothing more than the atrophied remains of the basement membrane of the tubes, with the healthy fibrous tissue arranged in the form of a net-work in which the tubes are packed, and which now appears more abundant in consequence of the wasting of the tubes.*

It has already been stated that the Malpighian bodies are unaffected in the progress of this disease, and this is true in so far as they remain for the most part free from any deposit or accumulation in their interior; but they must necessarily be affected by the changes occurring in other parts of the organ. Thus the destruction of many of the Malpighian bodies is a necessary consequence of the simultaneous wasting of the vessels and tubes which occurs in the advanced stages of chronic nephritis: and during the progress of the disease, the vessels of the Malpighian tuft will be in a state of more or less active congestion, in proportion to the rapidity of secretion and of cell-development in the tubes; and one consequence of this congestion of the Malpighian bodies will be the escape of serum into the tubes and the mixture of albuminous matter with the urine. The quantity of albumen in the urine will be great in proportion as the disease approaches in activity to that form which I have called "acute desquamative nephritis." When the disease is chronic and inactive there may be no albumen in the urine, or, it may be present in quantities so small as not to be detected by the ordinary chemical tests. In such cases, as indeed for the

* See Plate I. in the 29th vol. of the Med.-Chir. Transactions.

accurate discrimination of all forms of renal disease, the microscope will be found an invaluable aid. It must be remembered that the essential change in this disease is a destruction of the epithelial cells in the manner already described; the best evidence of this change being in progress is, the presence in the urine of moulds of the urinary tubes, composed of more or less disintegrated epithelium; and such evidence I have repeatedly obtained when no albumen could be detected by the ordinary heat and nitric acid tests.

A sufficient explanation has already been given of the small quantity of the saline constituents excreted by the kidneys in cases of chronic nephritis. It is manifest that if the epithelial cells are the agents by which the solid constituents of the urine are separated from the blood, a deficient excretion of these materials will be a necessary consequence of the greater or less destruction of the epithelial cells.

Another remarkable phenomenon in some of these cases admits, I believe, of an equally simple explanation: I allude to the large quantity of the watery part of the urine not unfrequently excreted by kidneys which are found after death to be very much wasted and contracted. It is by no means unusual to find patients with chronic nephritis passing 60 or 80 oz. of urine in 24 hours. What has already been said of the Malpighian bodies remaining healthy, and of the condition in which some of the tubes remain after being deprived of their epithelium, is a sufficient *anatomical* explanation of the fact, and the following appears to be the *physiological* explanation:—In health there is, within certain limits, a pretty constant relation between the quantity of solids excreted by the epithelial cells and the amount of water poured out by the Malpighian bodies. The relation is not, of course, very precise and constant, for, if it were so, the urine would invariably have the same density. There are many and various disturbing influences; but it may be stated, as a general rule, that the convoluted tubes and the Malpighian bodies act in concert, so that if more solid matter is excreted by the cells, a correspondingly increased amount of water is poured out from the Mal-

pighian bodies. In diabetes, for instance, the amount of water increases in proportion to the increase in the quantity of sugar. The cause of the increased flow of water in diabetes may be one of two conditions: 1st. It may be that the quantity of sugar *in the blood*, circulating through the Malpighian vessels, is the stimulus which excites these vessels to pour out water; or, 2nd, it may be the sugar in the *secreting cells* of the tubes which, as it were by a reflex action, excites the flow of water from the Malpighian vessels. Whichever of these two explanations be the correct one, the conditions for both exist in a case of chronic nephritis. If the presence of the solid urinary constituents in the blood be the cause of the phenomenon in question, it is well known that, as a consequence of renal disease, these materials are retained in such quantity as to be always injurious and frequently fatal to the patient. If, on the other hand, the Malpighian bodies are stimulated to secrete water by the presence, in the tubes, of the cells with their solid contents, it has already been shown that, as a consequence of chronic nephritis, these cells accumulate in the tubes in such abundance, that they may be supposed to excite the Malpighian bodies to action, in a manner not unlike the excitation of the lachrymal gland by a foreign body on the conjunctiva.*

Before leaving the subject of chronic inflammation of the kidney, I would refer to the case communicated to this Society by Mr. Busk, and published in the last volume of the Transactions, as a good instance of the form of disease under consideration. Mr. Busk is inclined to attribute the disease in that case to "the double duty the gland was called upon to perform," in consequence of the congenital deficiency of the other kidney. I should suppose the disease to have been the

* During an acute attack of nephritis, the aqueous portion of the urine is scanty on account of the vascular congestion and the consequent retardation of the circulation; but when the disease subsides and the congestion diminishes, the secretion often becomes abundant, and continues so until the renal tubes are thereby cleared of the morbid products which had accumulated within them.

result of the excretion, by the kidney, of abnormal and irritating products connected with the "occasional attacks of painful swelling of the foot, which were of short duration and considered to be of a gouty nature."

It must never be forgotten that these diseases are essentially connected with changes in the secreting cells; that, so long as the materials to be excreted are normal, the cells retain their healthy character, and an increased amount of normal excretion produces merely an increase in the size of the entire gland, without any change in the constitution of its several parts; while an unnatural condition of the excreted products is attended by some corresponding alteration in the character of the secreting cells.

Before concluding this communication on the inflammatory diseases of the kidney, it appears desirable to allude very briefly to the subject of my last paper:—viz. "Fatty Degeneration of the Kidney,"—my object being to show how essentially distinct are the two forms of disease; and at the same time to explain the manner in which they are sometimes combined. The observations which I have to make on this subject may be considered as an

APPENDIX

To a Paper on the Minute Anatomy and Pathology of Bright's Disease, &c., published in the Society's Transactions, Vol. xxix.

For some months past I have been aware that fatty degeneration of the kidney occurs in two distinct forms. The first form is represented in a drawing made for me by Mr. Brinton. The disease occurred in the person of Susan Smith, aet. 30, who died in the King's College Hospital, under the care of Dr. Budd, to whose kindness I am indebted for notes of the case. She had general dropsy, and her urine was very albuminous. The kidneys were in a state of "simple fatty degeneration;" they were large, smooth, soft and mottled, and were scattered over with

haemorrhagic spots. On a microscopical examination there was found no increase in the number of epithelial cells, nor inflammatory products of any kind, but simply a great accumulation of oil-globules in the epithelial cells; and consequently a filling and distention of the urinary tubules which contain the cells. The albuminous condition of the urine, and the haemorrhagic spots on the kidney, were doubtless the result of passive congestion of the Malpighian vessels, consequent on compression of the portal plexus by the distended urinary tubes.

In this simple fatty degeneration of the kidney all the tubes become almost uniformly distended with oil. In a slight degree and in the earlier stages, it is often found after death, in cases where there is no reason to suspect that it has been productive of any mischief during life: it is not until the fatty accumulation has attained a certain amount that the functions of the kidney are interfered with. It is this form of fatty degeneration of the kidney which occurs in animals, as a consequence of confinement in a dark room. In the human subject, although in the earlier stages it is a very common occurrence, yet in the more advanced stages it occurs less frequently than the *second form of fatty degeneration*. This form of the disease is represented in the 5th figure of Dr. Bright's 3rd Plate, as well as in the 1st, 2nd, 5th, and 6th figures of Rayer's 8th Plate. The cortical portion of the kidney, to use the words of Dr. Bright, is soft and pale, and interspersed with numerous small yellow opaque specks. The kidney is generally enlarged; sometimes it is even double the natural size. In some cases the cortical portion is somewhat atrophied and granular, but neither in this nor in the first form of fatty degeneration of the kidney does that extreme wasting with granulation occur, which is so frequent a consequence of chronic nephritis.

On a microscopical examination the convoluted tubes are found filled in different degrees with oil, some tubes being quite free while others are ruptured by the great accumulation in their interior. The opaque yellow spots scattered

throughout the kidney are neither more nor less than convoluted tubes distended, and many of them ruptured by their accumulated fatty contents ; just as the red spots are found to be convoluted tubes filled with blood. The cells which contain the oil are for the most part smaller, more transparent, and less irregular in their outline than the ordinary healthy epithelium ; they are increased in number, and many of them are so distended with oil as to appear quite black. In parts of the same kidney, there may commonly be seen some of the appearances already described as indicative of desquamative nephritis. This form of disease is very commonly combined with fatty degeneration of the liver, but less frequently than is the first form of fatty degeneration of the kidney.

It was not until very recently that I could give an explanation of the appearances here described, and of the difference between this and the first form of fat kidney. At present I have under my care, at the Public Dispensary, a patient, the progress of whose case has afforded me the light I had for some time been in search of. I will give so much of this man's history as will suffice to render my explanation intelligible.

John St. Ledger, æt. 49, a billiard-marker, until the commencement of the present year has always led a very irregular and intemperate life. About eight years ago he had an attack of general dropsy, with very scanty and high-coloured urine. He was bled from the arm ; and in about six weeks he recovered completely. He remained well until about four years ago, when he had a similar attack from which he recovered in about a month : from this time he continued well until the commencement of the present illness, which he thus accounts for :—During the period of Lent in the present year he fasted, i. e. during four days in the week he took only one meal a-day ; this meal was composed of fish with milk and potatoes ; during the remaining three days he took animal food as usual, perhaps rather in excess. Under this mode of living he found himself becoming thin

and weak, and towards the end of Lent he had lost flesh considerably, and was very dyspeptic and flatulent after his meals. On Easter Monday, April 5th, he felt drowsy and had muscular pains; the next day he had dropsical swelling, and the urine was very scanty. He continued to get worse until Monday, April 12th, when he applied at the Dispensary. There was then slight general dropsy, headache and drowsiness; the urine was scanty, high-coloured and very albuminous. On a microscopical examination I found in it all the appearances which are characteristic of simple acute desquamative nephritis—i. e. there were blood-corpuses, fibrinous casts of the tubes, and epithelial cells in great numbers. During the first few days he was much benefited by cupping, warm-baths, purging and diaphoretics.

A careful microscopical examination of the urine was made almost daily; the characters remained the same until April 29th, when it was found that many of the epithelial cells contained a considerable number of oil-globules; and from that time to the present he has continually passed fibrinous casts and epithelial cells, many of which are completely distended with oil-globules; and in proportion as they become filled with oil they appear to lose their angular outline, and become transparent and globular or oval. At the present time the urine is highly albuminous, and the poor man is gradually becoming more pallid and emaciated.

Here then is an explanation of the second form of fatty degeneration of the kidney, the peculiarities of which result from a nephritic condition of the organ, dependent on the presence of some irritating material in the blood being associated with a tendency to fatty degeneration; this tendency resulting from the presence in the blood of mal-assimilated fatty matter. The nephritic condition is manifested by an increase in the number of epithelial cells; the tendency to fatty degeneration, by a filling of many of these with oil. Although the two conditions are combined in this and in similar cases, it must be remembered that they are essentially distinct in their nature and origin. Each cell which escapes

from the kidney carries with it a portion of the morbid material. The oil is in the form of visible globules, while the cells which contain no oil doubtless contain some other material which is invisible, or less readily seen than the oil-globules.

That the oil is an excretion, is to a certain extent proved by an observation which I have recently made in the case of a girl named Amelia Robinson, at present in King's College Hospital under the care of Dr. Todd. This patient has dropsy with albuminous urine, dependent on acute nephritis; the very disordered state of her nutrition is clearly shown by the materials contained in her urine. These are, in addition to albumen and blood, triple phosphates, phosphate of lime and oxalate of lime. There are many globular and oval cells exactly similar to those which are commonly found to contain oil, filled in different degrees with the octahedral crystals of oxalate of lime. (See Plate VI. fig. 9.) It cannot be doubted that the oxalate of lime is excreted by these cells, and that its presence in the urine is a consequence of disordered general nutrition, and not of any local disease in the kidney; and there is as little reason to doubt that the presence of oil in the urine is the result of an analogous disorder.

I have now distinguished and described four conditions of the kidney :

- 1st. Acute desquamative nephritis.
- 2nd. Chronic desquamative nephritis.
- 3rd. Simple fatty degeneration, and
- 4th. A combination of fatty degeneration with desquamative nephritis.*

In all these diseases the morbid materials are deposited in the urinary tubules, from which portions of them become

* The two forms of inflammatory disease above mentioned are not the only diseases of an inflammatory nature to which the kidney is liable as a consequence of constitutional disorder. The author has notes of cases, in some of which the morbid material deposited in the kidney and eliminated with the urine has been chiefly pus, while in another and a distinct class of cases it has been chiefly blood. As an example of the latter

washed out, and thus mingled with the secretion. The diagnosis of each of these conditions of the kidney, during the life of the patient, is a matter of the greatest importance with reference to prognosis and treatment, and the diagnosis may be made with ease and certainty by a microscopical examination of the urine.

It must be remembered that the materials in question are all, with the exception of free oil-globules, of greater density than the urine, so that in a short time they fall to the bottom of the vessel in which the fluid is contained. In collecting the urine for examination, care must be taken to obtain the sedimentary portion; the upper part of the liquid may be poured off, and the last three or four ounces should be shaken up and taken for examination.

When the quantity of material is small, as often happens in cases of chronic nephritis, the examination is much facilitated by placing the urine in a conical glass, shaped like an ale-glass; after standing a short time, the material we are in search of collects within a small space at the bottom of the vessel: with a pipette a few drops may be taken from the lower part of the liquid, placed in a small cell, then covered with a thin plate of glass and examined with a power of about 200 diameters. In the majority of cases an examination occupying only two or three minutes will afford information, as to the condition of the kidney, not less certain nor less valuable than that obtained by a physical examination of the chest in cases of pulmonary and cardiac disease.

If the account here given of these diseases be a faithful and a correct one, without doubt it throws great light, not merely on their pathology but also on their treatment. It teaches us that these morbid conditions of the kidney do not

form of disease he would mention cases of haematuria induced by the administration of oil of turpentine or cantharides; and he has observed several cases in which the exciting cause has been of a different nature. He will take an early opportunity of bringing these observations before the public, and hopes to show that he does not over-estimate their pathological and practical importance.

originate in the organ itself, but rather that they are the local manifestations of a more general constitutional disorder.

The pathological changes which the kidney undergoes are such as it is liable to in the performance of its physiological duty, that, namely, of separating from the blood materials, the retention of which would interfere with the normal and healthy condition of that fluid.

In the treatment of these diseases two indications must be kept constantly in view. These are, first,—To prevent the further formation or development of those products, the excretion of which by the kidney is productive of serious structural changes; and second,—To relieve the kidney as much as may be of its excretory duty, by exciting to action other eliminating organs—as, for instance, the skin and bowels.

With reference to the mutual connection between the skin and the kidneys, the following general principles appear worthy of attention:—First, When the kidneys are suffering in consequence of having to excrete abnormal products, we may often with safety and advantage relieve these organs by exciting the skin to an increased amount of excretory work. On the contrary, when, in consequence of an abnormal condition of the blood, the skin is the seat of disease, the greatest care and caution are required in the administration of cantharides and other diuretic medicines; the danger being, that the *materies morbi* may by these means be determined to the kidneys, and that the cure of a trifling cutaneous disease may be accomplished at the risk of exciting a serious and destructive renal disease.

Second,—When cerebral disease supervenes upon the suppression of a cutaneous eruption, it is probable that this occurs through the intervention of renal disease, and not as a consequence of the direct transfer of disease from the skin to the brain or its membranes.

It has been too much the custom to administer medicines for the purpose of stimulating the kidneys, in all cases in which these organs are supposed to be in a state of functional inactivity. There are certain materials derived both from

the organic and the inorganic kingdom, the administration of which to a person in health is constantly followed by an increased flow of urine, and at the first view of the matter, it appears natural to suppose that the same materials would be equally efficacious in increasing the quantity of urine when the secretion has become scanty in consequence of renal disease. A very small amount of physiological knowledge, however, is sufficient to explain the universally acknowledged uncertainty, or rather the almost certain want of success, attending the administration of diuretic remedies in all cases of renal disease. The most certain diuretics are the normal solid constituents of the urine—experiment sufficiently shows this. Dr. Todd relates (in a clinical lecture published in the *Medical Gazette* for 1845) that “he once injected half a drachm of urea into the vein of a dog, and the only effect produced was an excessive secretion of urine. The place where the dog was kept was literally flooded in an hour or two by the frequency and quantity of his micturition.” In almost every case of renal disease these natural diuretics accumulate in the blood, not on account of any mere functional inactivity unconnected with organic change, but because the secreting structure of the kidney is more or less completely destroyed. Under such circumstances, the administration of medicines which would be excreted by a healthy kidney is like adding to the load on a porter’s back, since it increases the demand upon the labour of the kidney, without in any degree adding to its powers of performance. There is reason to believe that in cases of this kind, and more especially perhaps in cases of chronic nephritis, the only safe and useful diuretic is pure water.

EXPLANATION OF PLATE VI.

This plate illustrates Dr. Johnson's paper on the Inflammatory Diseases of the Kidney.

Fig. 1. Section of a portion of inflamed kidney. The tubes appear as if divided into distinct globular and oval portions; this appearance results from the manner in which the tubes are packed in the meshes of the fibrous tissue, so as to be concealed where they are crossed by the fibrous tissue, and visible in the intervals. The tubes are rendered opaque by an accumulation of epithelium, the outline of the cells being invisible on account of their crowded condition. A Malpighian body in the centre of the mass appears transparent and healthy. (See p. 175. Magnified 200 diameters.)

Fig. 2. Section of a portion of kidney, showing the tubes deprived of their epithelium by "chronic desquamative nephritis." The appearance of the tubes is very similar to that of globular and oval transparent cysts; the explanation of this appearance is given above. (See p. 175. Magnified 200 diameters.)

Fig. 3. Section of a portion of kidney, showing the tubes lined by delicate transparent cells. (See p. 175. Magnified 200 diameters.)

Fig. 4. Portion of the basement membrane of a tube deprived of its epithelium, and contracted by its elasticity into an irregular globular form after being detached from the surrounding tissues. (Magnified 200 diameters.)

Fig. 5. Portion of a tube much dilated, and bulging in the intervals of the fibrous tissue: the cut extremity of the tube is seen at *a*. (Page 177. Magnified 200 diameters.)

Fig. 6. Portion of a tube much dilated, and divided by septa which correspond with the rings of fibrous tissue in the microscopic specimens. From preparation No. 871 in the King's College Museum; natural size.



Fig. 7. Includes, *a a*, fibrinous casts of the urinary tubes entangling epithelial cells and blood-corpuscles; *b b*, free epithelial cells; and *c c c*, crystals of lithic acid from the urine in a case of "acute desquamative nephritis." (Page 168. Magnified 200 diameters.)

Fig. 8. A mass of oily matter from the urine in a case of "chronic desquamative nephritis." (Page 177. Magnified 200 diameters.)

Fig. 9. A cell containing octohedral crystals of oxalate of lime from the urine. (Page 186. Magnified 400 diameters.)

Figs. 10, 11 and 12. Casts of the urinary tubes, composed of fibrinous matter and disintegrated epithelium from the urine, in a case of "chronic desquamative nephritis." (Magnified 200 diameters.)

For a note, having reference to the foregoing papers of Mr. Simon and Dr. Johnson, the reader is referred to the close of the volume.

AN ACCOUNT
OF THE
STRUCTURE OF A NÆVUS.

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COMMUNICATED BY T. B. CURLING.

Received May 11th—Read June 22nd, 1847.

THE nævus, the minute structure of which I am about to describe, was given to me by my friend Mr. Curling. It was situated upon the back of the hand of a young man, just below the carpal extremity of the ulna. The man stated that it had existed from birth. It was about the size of half-a-crown, soft, and of a bluish tint. In its centre the skin was thin. The veins on the back of the hand were rather larger than usual. It belonged to that class of nævi termed subcutaneous.

The entire mass of the abnormal growth was removed by excision, and the bleeding was very trifling. Two small arteries required ligatures.

The following elements composed the growth, viz.:

1. Areolar, uniting, or fibrous tissue.
2. Epithelium.
3. Capillary vessels and vessels of larger calibre.

1. *Of the Fibrous Tissue.*

The mass, before careful dissection, presented an irregular growth, apparently lobulated, consisting of fibrous tissue

which contained fat. When the fat and the uniting tissue were dissected off, several lobes, to the number of twelve, were recognisable. These lobes varied in size and in length, from between an eighth and three-quarters of an inch, and were attached by a kind of neck to the corium, but they were perfectly free upon all other sides.

Each lobe possessed a distinct and proper capsule, which at the neck became intimately blended with the fibrous tissue of the true skin.

The lobes, externally, resembled fibrous sacs depending from the corium, but both transverse and longitudinal sections exhibited clearly the internal structure of each. Short, thick, flat, delicate and strong fibrous bands intersected each in every direction without much regularity, although in some of the lobes a somewhat characteristic arrangement was discernible. In these a dense and well-defined central point existed, from which the bands or septa passed off, in a radiating manner, to the envelope or proper investing sheath.

Having encircled the neck of one of these lobes with a ligature, I inserted a tube and succeeded in distending it with air. The inflated lobe was removed and dried. Transverse sections exhibited very well the reticular character of the interior. Sections of other undried lobes display these cells, meshes or interstices very clearly. The cells communicate with one another on all sides, and they vary greatly in their size. They open at their peripheral extremity into large spaces, or what might be termed *reservoirs*, in the corium. At this point, however, the different lobes forming the abnormal growth do not all appear to communicate with each other.

2. *Of the Septa or Bands, or Lamineæ.*

The septa vary considerably in diameter. They are composed of a most delicate fibre tissue, wavy and regular. A particular kind of epithelium is arranged over their surface, being clear and well-marked in almost every place. The

arrangement becomes more defined after the action of dilute acetic acid. The epithelium is of the class termed pavement or tessellated, and the nuclei are remarkably distinct.

Sometimes it appears as if the epithelium were supported by a very delicate structureless membrane (basement membrane of Bowman?).

The nucleus fibres of Henle, the yellow fibrous element of other authors, are found scattered and mingled with the tissue of the bands.

Vascular Arrangement.

I was unable to inject the specimen from the small arteries supplying it.

At the time that I was dissecting the cellular tissue from between the lobes I carefully preserved several small blood-vessels, which I followed into the fibrous envelope of the separate lobes. They could not be traced into the septa, nor did they appear to open into the cells, although, perhaps, both these arrangements exist.

Again, upon dividing that portion of the proper envelope of the lobe, which, at its neck, intimately blends with the corium, a large space, or, as I have before termed it, reservoir, is seen, through which delicate filaments traverse from the corium to the septa of the lobe. Minute examination proves that these are delicate vessels, somewhat looped, and in many instances they were distended with blood-corpuscles. They appeared to be lost upon the septa.

These reservoirs communicate with veins, which are usually found more or less distended in the proximity of the abnormal growth.

Each lobe appeared to possess two or three small arteries. I did not find one vessel supplying two lobes, but each lobe received its own distinct vessel or vessels. These vessels presented the characteristic appearances of the vascular tissues, when acted upon by acetic acid and examined by a magnifying power.

Histological Relations.

The various names given to these tumours, and the resemblances which they are said to afford to normal tissues of the body, oblige me to state my opinion in regard to their histological affinities.

1. There is no more reason to give them the title of 'vascular or blood-vessel tumours' than many others which have received very different names.
2. They consist neither of small arteries, of small veins, nor of a mixture of these vessels,—tumours thus formed having their more appropriate names—aneurism by anastomosis, varicose veins.

3. They do not consist of erectile tissue, this term being understood to refer to such tissues as the *Corp. cavernosum, penis, &c.*, because they do not present the appearances exhibited by these tissues under the microscope, the supposed *Art. Helicinae* of Müller, the unstriped muscular fibres according to Valentin, or the clear and distinct arterial capillary ramifications as described by Henle and Müller. Besides, all the cells communicate with each other within the lobes, an arrangement which cannot possibly exist in the *Corp. Cavernosum* and similar structures. In these there must be two sets of cells, one set which may be termed the venous from their connection with the veins, and another set which probably have no natural communications with vessels, but which, in the event of a blood-vessel giving way, receive the extravasated blood, and, becoming distended, give rise to permanent erection of the intromittent organ. However, they more closely resemble the *Corp. cavernosum* than any other tissue.

4. They must be classed with the fibrous tissues, being developed probably like them, nourished by arteries which may differ greatly in size, and possessing cells which are in communication with larger or smaller veins. Rokitansky says that he has seen a tumour of this kind in communication with the saphena vein of the thigh, and there is a case

on record of a similar growth which was connected with the external jugular vein.

Practical Considerations.

If it be admitted that the description here given pertains to all, or, at least most, *true* subcutaneous nævi, the course of treatment to be pursued cannot fail of being tolerably clearly indicated.

In those cases in which the arteries supplying the lobes are large and their pulsation can be distinctly felt, the application of a ligature to the arterial trunk from which they come off may be required.

Their structure clearly indicates the necessity of *total* and *complete* excision, when this proceeding is determined upon; although I believe that the large openings described and regarded as veins, are not, in fact, such vessels, but merely an appearance produced by a section of one of the reticulated lobes.

This case being regarded as typical of these growths, it points to the necessity of adopting such treatment as shall effectually operate upon the entire mass, and not upon one or two of the lobes only.

The intimate communication subsisting between the reticular texture of the lobe and the veins, which are often very large, points to the danger which may result from injecting these growths with stimulating fluids.

The nature of the new growth being essentially fibrous, the great desideratum is the production, within its meshes, of such a degree of adhesive inflammation as will serve to obstruct the entrance of blood or its passage from one cell to another; or, the employment of such means as will, by creating greater disturbance, give rise to obliteration of the cells, or, indeed, procure destruction of the entire growth by suppurative inflammation.

The treatment of *nævus maternus*, as recommended and practised from the time of J. L. Petit to the present day, resolves itself into the three following methods:—

1. Entire removal, by excision ; or the adoption of such means as shall arrest the supply of blood, and produce a complete *sloughing* away of the abnormal growth or its destruction by ulceration.
2. The employment of such substances as will give rise to *suppurative* inflammation.
3. The production of *adhesive* inflammation within the lobes, and consequent obliteration of the reticulated interior.

A CASE
IN WHICH
A LARGE POUCH WAS FORMED IN THE
ŒSOPHAGUS,
IN CONNECTION WITH
CONTRACTION OF THE CANAL.

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Received May 24th—Read June 22nd, 1847.

JUDGING from the circumstance of so few cases having been furnished by medical authorities, the formation of a sac or pouch in the œsophagus, in connection with stricture of this canal, is of rare occurrence. This lesion, however, has been noticed by Isenflamm, Sandifort, Meckel, Copland and Rokitansky. Two cases, in which it was found after death, are also described by Mr. Ludlow of Bristol, and by Sir Charles Bell.

The case observed by Mr. Ludlow was published in the 3rd volume of *Medical Observations and Enquiries*, in 1764. The subject of this case was a man aged 60 years, who attributed the commencement of his disease to a cherry-stone which had lodged in his throat about five years before his death; dysphagia, &c., gradually supervening. Upon examination after death, a muscular bag was seen passing down between the œsophagus and vertebrae. The inferior point of this bag reached some little way into the cavity of the thorax,

hanging quite loose and situated principally on the right side. A probang could be made to enter easily along the œsophagus; and, by giving the point of the instrument a turn forwards, to pass readily into the stomach. The bag was stated to have been formed by a dilatation of the entire substance of the posterior part of the pharynx, the structure being so equal that it was impossible to ascertain at what particular part the dilatation began. Mr. Ludlow has entered into a full explanation of the reasons wherefore this dilatation had taken place whilst the passage along the œsophagus still remained; and has shown that, the opening into this sac having been wider than that into the œsophagus, no food could pass into the latter until the former was filled: but I must refer to the case itself, and to the plates given with it, for further details.

A somewhat similar case to the foregoing is described by Sir C. Bell, in the second volume of his *Surgical Observations*. A gentleman applied for the relief of a disease of the throat, occasioning difficulty of deglutition. Many inefficient attempts were made to pass a bougie. In carrying the point towards the back of the pharynx, the obstruction was so great that the operator would not venture to press with force. By giving the instrument a turn laterally, the point was brought to pass by the side of the throat, and moved along the lateral part of the pharynx down to the proper canal. It was observed that when the bougie was introduced it was not grasped, nor was there any impression on the soft wax. The operation was repeated at intervals of a fortnight, and the symptoms were always relieved by the passing of the bougie. The patient's death does not appear to have been caused by the complaint in his throat, but by what cause is not stated.

Upon examination after death, a bag was found projecting from the lower and posterior part of the pharynx, and pushed into a space between the œsophagus and spine. The bag was not covered with muscular fibres, but was described as a hernia or protrusion of the inner coat of the pharynx between the fibres of the muscular coat.

Sir C. Bell concludes by adverting to Mr. Ludlow's case ; and, with an ingenuity which ever distinguished that great surgeon, proceeds to reason upon the two cases. His opinion is at variance with that of Mr. Ludlow as to the precise nature of the lesion : for he does not consider the sac to have been muscular in that case, but to have been caused, as in the case described by himself, by the protrusion of the inner coat of the pharynx through the fasciculi of the constrictor pharyngeus. This he believes might have been occasioned by the repeated and ineffectual gulping, or efforts to swallow. I now proceed to state the following case :—

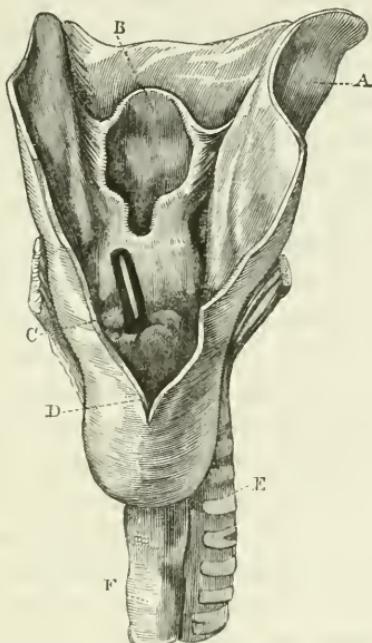
Col. D—, aged 69 years, of a robust constitution, indulged during the greater part of his life a very great appetite. Three years before his death he experienced slight dysphagia, which continued without any increase for eighteen months, his general health continuing unimpaired. During this period he consulted an eminent practitioner in London, who, judging from his excellent state of health, looked upon his symptoms as arising merely from a slight spasmodic affection, and dismissed him with the impression that no serious disease existed. No restrictions having been imposed upon him as to diet, he continued to indulge his appetite as usual. In January and February 1846 deglutition became more and more impeded, and was accompanied by emaciation ; but although he was residing in London at that period he did not have recourse to medical advice. In July he visited Lowestoft, and I was consulted. There was then great difficulty of swallowing, especially the more solid articles, which required the most complete mastication before they could pass into the oesophagus, the passage being attended by a gulping noise. A fluid, resembling saliva, was constantly secreted, and frequently voided from the mouth to the amount of a pint or a pint and a half in the twenty-four hours ; and was occasionally mixed with food. As a consultation was proposed with Mr. Crosse, of Norwich, I deferred an examination until that gentleman should be present. A probang was first introduced, the point of which was resisted below the

cricoid cartilage. A slender wax bougie was next employed, which Mr. Crosse conceived had advanced beyond the apprehended seat of stricture; but this admitted of doubt, owing to the appearance of the instrument when withdrawn. Subsequent attempts to pass it beyond the apparent seat of obstruction proved abortive.

From this time the patient lingered on, emaciation increasing. Notwithstanding injunctions to adopt a liquid diet, he daily persisted in the use of solids. It was frequently remarked that during a meal, a portion of food appeared to be swallowed and for a time retained, but was shortly afterwards returned very little changed; regurgitation taking place in a way similar to the rumination of animals. Eventually the patient became incapable of receiving any food into his stomach. Three weeks previously to death, which took place on the 21st October, he was sustained solely by a pint of strong beef-tea, containing a glass of sherry, injected into the lower bowels every eight hours.

Upon examining the body after death, the pharynx, oesophagus, larynx and trachea were carefully removed for closer inspection. The parts being cleared of cellular substance, a bag or pouch was discovered projecting from behind the oesophagus, opposite the cricoid cartilage, and hanging down between the trachea and oesophagus and the cervical vertebrae; it measured three inches and a half in length and two and a half in circumference, and was in shape not unlike the finger of a glove. Nearly two-thirds of this pouch were covered by muscular fasciculi derived from the pharyngeal constrictors, the fibres of which were more developed than in health. The pharynx was laid open by an incision commencing at its upper and posterior border, in the direction of the median line, and continued into the pouch two-thirds of its length. This exposed the entire pharyngeal cavity, which was dilated very far beyond what was natural. The opening from one cavity into the other was perfectly free; and the two, when expanded, presented a funnel-like appearance, capable of holding nearly two pints of fluid. Immediately behind the cricoid cartilage,

and on a level with the commencement of the pouch, existed a stricture, formed by a transverse fold of the mucous membrane, that would admit only a large-sized urethral bougie. The oesophagus below the stricture was much contracted, but the mucous surface was perfectly healthy.



This wood-cut represents the posterior view of the pharynx, showing the situation of the sac.

A. Pharynx very much dilated. B. Epiglottis. C. Stricture shown by the introduction of a bougie. D. The sac laid open. E. Trachea. F. Oesophagus.

After considering the history of this case, the following questions suggest themselves:—1st. How may cases, similar to or identical with the one now related, be recognised with tolerable accuracy during the life of the patient? And 2ndly. What is the best mode of treating them? It may be remarked, with reference to the first question, that cases of this kind occur so seldom, that a correct diagnosis is formed with difficulty; and the difficulty will exist until the early history

of similar cases is correctly observed and recorded. It may be remarked, that in neither of the two cases published by Mr. Ludlow and Sir C. Bell did any stricture exist, although the patients were treated for stricture. The pouches found after death were entirely unlooked for. The formation of pouches or sacs, such as were found in the present case and in those referred to, is most likely to be caused by stricture, and therefore to be ascribed to, and confounded with, that lesion. The existence, however, of a stricture may be readily inferred from symptoms; and, when existing simply, is easily ascertained. But the presence of a pouch, such as was found in the former cases, may prevent the existence of stricture from being ascertained during life, as occurred in respect of them; and the co-existence of a pouch with stricture may suggest the latter lesion without the recognition of the former. There is, however, one symptom which was present both in Mr. Ludlow's case and my own, which may assist in the diagnosis; and this was the peculiar way in which the food was regurgitated after having been apparently swallowed; a slow regurgitation of it taking place at various periods after it had been taken. In an ordinary case of stricture, the patient is generally unable to swallow more consistent matters; or, if the attempt be made, they return immediately, attended by a distressing sensation of suffocation. Columbat's speculum might be made available in cases similar to the present; for an ulcer seated at the rima glottidis may be brought into view by its assistance.

As respects treatment, I am not prepared to suggest any means worthy of attention. If, however, a similar case to the one now stated were to come under my care, or one in which the existence of a pouch was ascertained, I should act as much as possible according to the suggestion of Sir C. Bell, and endeavour to introduce a tube into the oesophagus, through which the patient might be fed, so as to prevent the passage of any food into the pouch. The practicability, also, of syringing the pouch with astringent injections might be entertained.

The formation of the pouch in the present case may be easily explained. As soon as an obstruction to the free passage of food along the oesophagus commenced, the action of the pharyngeal muscles, propelling the alimentary bolus against the stricture, distended the part intervening between these muscles and the stricture; and as the disention increased, one portion of the parietes of the part yielded more than the rest, and at last formed the pouch found on dissection. Had the stricture been discovered at an earlier period of its formation, it is more than probable that the disease might have been arrested or greatly palliated by a judicious employment of bougies; for, upon examination, it did not appear that any degeneration of structure existed.

ON
THE CONTRACTILITY
OR
IRRITABILITY OF THE MUSCLES OF
PARALYSED LIMBS,

AND THEIR EXCITABILITY BY THE GALVANIC CURRENT,
IN COMPARISON WITH THE CORRESPONDING MUSCLES
OF HEALTHY LIMBS.

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THIS subject has already been brought under the notice of the Fellows of this Society by Dr. Marshall Hall, who has put forward his views respecting it in a paper printed in the 22nd volume of the Transactions. The statements made in that paper have been repeated by its author in the article "Irritability," in the Cyclopaedia of Anatomy and Physiology, and subsequently in a volume of Essays very recently published.*

In these various writings Dr. Marshall Hall makes a distinction between the paralysis which results from lesion of the spinal cord and that produced by lesion of the brain. The distinction laid down by him may be thus expressed:—In cerebral paralysis, the irritability of the affected muscles becomes augmented: in spinal paralysis, the muscles supplied with nerves from the diseased portion of the spinal cord quickly

* See also Essay on the True Spinal Marrow, &c., 1837, p. 102.

lose their irritability; and the same effect is produced in paralysis dependent on disease of the principal muscular nerve of a limb or other segment of the body.

It must be confessed that this doctrine, so far as regards *cerebral paralysis*, is not a little startling to those who have been accustomed to admit, what experience so amply justifies, that the healthy action of a muscle is promoted by exercise within reasonable limits; and who would naturally infer that whatever restricted that exercise would be necessarily injurious to the nutrition of the muscle, and consequently to its leading vital property, namely, its irritability.

It would seem, from the perusal of Dr. M. Hall's writings upon this subject, that this doctrine suggested itself to him in reflecting on the curious fact noticed first by Fouquier, and now familiar to all physicians, namely, that during the administration of strychnine in certain cases of paralysis, the peculiar spasms which that drug is capable of exciting are first observed in the paralytic muscles. Referring to this fact, as regards the influence of strychnine, Dr. M. Hall makes the following observations:—"It will soon be seen that this view, like a former one, is far too general—far too indiscriminate; that it is not in every case of paralysis that the strychnine would first display its influence on the paralytic limbs. Meantime, however, I figured to myself the fact of the strychnine acting on the spinal marrow, and diffusing its power equally along the nerves, to the right hand and to the left, to the muscles to which they proceed respectively: and I asked myself the question—Is the difference observed in its ultimate effects on those muscles, the power being obviously the same, owing to a difference in the degree of the irritability of the muscular fibre itself? Is the irritability of that fibre actually augmented? if so, the phenomenon would be explained."

The results of experiments, instituted by Dr. Hall in order to solve this problem, confirmed his views, and led to the adoption of the opinion, that the abstraction of the influence of the brain upon a limb permitted an augmentation of

the irritability of its muscles; and this, again, gave confirmation to the hypothesis that the spinal cord was the source of muscular irritability; and that the ordinary actions of the brain served to exhaust this irritability, which, of course, would accumulate in the muscles, when the exhausting power of the brain had been withdrawn through the influence of disease.*

Feeling much dissatisfied with these views, which appeared to me to be inconsistent with many ascertained facts in the physiological history of the brain and spinal cord, I determined to repeat the experiments whenever opportunity offered, and I now venture to lay the results of my observations before the Society.

I may here state, that most of my experiments were made in hospital practice, and in presence of a large number of students, who generally attended in greater numbers when they knew that something experimental or operative was to be shown.

In the experiments the electro-dynamic machine was used, which was supplied with a single cell of Daniell's constant battery. Latterly I have employed the magneto-electric rotation machine, which does not require a battery, and which is on that account peculiarly convenient for medical purposes. It is very easy to regulate the rapidity and violence of the shocks. Only rarely have I used the galvanic battery alone; the use of this mode of developing the electric current is difficult in practice, in consequence of the trouble necessary to be taken to prepare the battery.

A little consideration will, I think, indicate that the results of the experiments need not be in any way affected by the instrument employed. The electro-dynamic apparatus is but

* "We may further deduce," says Dr. Hall, "from the facts which have been detailed, that the spinal marrow, and not the cerebrum, is the special source of the power in the nerves of exciting contraction, and of the irritability of the muscular fibre; that the cerebrum is, on the contrary, the exhauster, through its acts of volition, of the muscular irritability." Med.-Chir. Trans., vol. xxii. p. 205.

a substitute for a large battery; the electricity generated in a single cell passes along a coil of wire of considerable length, and induces another current in an adjacent coil, which is itself also of great length. This current may be again augmented by placing a bar of soft iron, or, still better, a bundle of iron wires, at right angles to the coils. The battery is connected with the primary coil, and, by means of a rotatory wheel, contact may be easily made or broken: the intensity of each shock will, of course, depend on the extent of the *inductric* and *inductiveous* material (to use Dr. Faraday's terms): the physiological effects, however, will be very much influenced by the rapidity with which the shocks are made to succeed each other, and this may be regulated by the rate of rotation of the wheel.

Of similar kind is that most convenient instrument the magneto-electric rotation machine. The electricity is obtained by induction from a powerful magnet, and great convenience is afforded by its being always ready for use and not needing the troublesome appendage of a battery.

In most of the experiments, the limbs to be compared were immersed by the hands, or the feet, as the case might be, each in a basin of water, which was connected with one of the wires of the galvanic apparatus. The current thus passed through both limbs at once, and both were similarly and simultaneously exposed to its influence.

I select from my case-books several cases in which I have tested the relative irritability of the paralysed and sound limbs by the methods above indicated.

Case 1.—Jane Weedon, æt. 61, hemiplegia of the right side—the paralysis of the arm is complete, that of the leg incomplete—motion only is affected in both limbs, sensibility being perfect. The attack was sudden; on getting out of bed in the morning she was seized with a violent fit of coughing, and staggered to her bed; she immediately lost her speech, retaining, however, her senses. In an hour she regained her articulation, was cupped and blistered, but seven hours afterwards lost her speech again, and with it the power of the right

arm, and that of the right leg but in a less degree. Her intellect and memory were slightly impaired.

This was a hard-working washerwoman, who had to support and bring up a large family by her own industry, her husband having been some years dead. I diagnosed the case as one of white softening, with perhaps a slight apoplectic clot or clots affecting the left hemisphere of the brain, and involving some of the fibres of the corpus striatum.

As the paralysis of motion was complete in the arm, the cerebral influence being completely cut off from that limb, the case was a fair one for applying the galvanic test to the muscles of the healthy and palsied arms.

The galvanic current was passed through the arms on the 10th May 1845, the hands being placed in basins, as already described. The electro-dynamic machine was employed—at first weak, without the bundle of wires, which is used to augment the current, the wheel being made to revolve slowly so that the shocks succeeded each other at long intervals.

By the weak current *no contraction of the muscles* was produced in the paralysed arm—slight contractions in the sound one. By the stronger current no contractions were aroused in the muscles of the paralysed arm, until the stimulus had been continued for some time, when only very slight contractions were excited, which were trifling when compared with those excited in the sound arms.

This patient remained under treatment for more than a month. She acquired much more power of her leg but none of the arm. On the 27th of July she returned to the hospital. The muscles of the right arm had wasted considerably: the palsy of that member had led to its disuse, which ought to have augmented the irritability of its muscles, if, as Dr. Hall affirms, “the constant *use* of the paralytic limb *diminishes* its irritability.” (Observations on Medicine, chap. xix. p. 118.)

The application of the electricity, however, on this occasion led to the same result as before, namely, very slight contractions of the palsied limb, and distinct and vigorous ones of the sound

limb. When the current was applied directly to the biceps muscle of each limb, that on the sound side contracted much more forcibly than the paralysed muscle.

I am glad to quote first a case in which the paralysis affected the arm; for Dr. M. Hall justly remarks, "The most unequivocal experiment is that made on the arms; because the paralytic seizure is in general most displayed in this part." (*Loc. cit.*, p. 123.)

Case 2.—This is a very similar case to the preceding one. Maryanne Twist, *aet.* 44, married, and had eight children. The attack was sudden, preceded by giddiness followed by paralysis of the left arm and leg, the former being completely palsied, the latter partially so. There were also paralysis of the tongue on the left side, as shown by its deviation to that side in its protrusion, and paralysis of the buccinator muscle of the cheek, which flaps somewhat in speaking.

The effects of the galvanism were the same as in the previous case, as far as regards the contraction of the muscles: it is interesting to notice that, in passing the current, more pain was felt in the palsied side than in the sound one.

Case 3.—James Stallard, *aet.* 54, a painter, and of intemperate habits. This was hemiplegia of the left side, affecting the face, arm and leg: the arm was completely paralysed and hung lifeless by his side: the leg was only partially paralysed: the attack was sudden, without loss of consciousness, and without warning: sensation was not affected. The man had for some time before the attack been living poorly. There was no infection of lead. I viewed the case as the result of white softening of the brain.

The result of the galvanic trials were, that while contractions were excited in both arms, those of the left or paralysed arm were obviously considerably less than those of the right or sound limb.

Case 4.—Henry Angus, *aet.* 61. Hemiplegia of the left side, especially the arm—sensation being very much impaired; the attack sudden, and much of the same character as in the previous case.

In this case, likewise, although the galvanic current excited the muscles of both limbs, those of the sound limb were much more strongly contracted than those of the paralysed limb.

Case 5.—Edward Woof, aet. 63. Hemiplegia of left side. The arm, leg, and side of the face were completely palsied as far as regards motor power. The attack was sudden, preceded however by a noise in the head, referred to the back part, “as if an engine had been at work in it.”

The galvanism was applied to this man’s arms. The palsied limb was very little affected by it, while the sound limb was made to contract strongly.

Case 6.—Caroline Willis, aet. 25. This patient had very complete hemiplegia of the right arm and leg, and also of the face. The arm was perfectly powerless; the leg was also similarly affected but to a less degree; she dragged it after her in walking. The face was perfectly motionless on the right side, the buccinator muscle being completely paralysed; and the orbicular muscle of the eyelids was slightly affected, as the patient was unable to close the eyelids on that side completely. The muscles of the paralysed limbs were flaccid and very soft.

From the history of this case, it appeared that there was some syphilitic taint in the system. The palsy was preceded by pain in the forehead and vertex, with drowsiness, general debility, and profuse leucorrhæal discharge, for which she had been inordinately bled before her admission. After the bleeding, her drowsiness increased and her intellect seemed impaired, and she had a violent hysterical fit which lasted half an hour. From this time her mother noticed some impediment in her speech, and occasional startings of the limbs. This state lasted about three weeks, when one day she suddenly lost the use of one side without any impairment of consciousness. Both sensation and motion were affected at first, but after a few days the sensibility was restored.

I viewed the case as one of white softening of the brain, preceded by some meningeal disease which probably was syphilitic in its nature.

The report of the trial of galvanism four days after her admission into the hospital is as follows:—"The current was passed through both arms in the usual way. Contractions took place in both limbs in a very manifest way. The action of the muscles on the paralysed side was distinctly less than that of the healthy ones, especially of the biceps muscle, and the paralysed arm was not jerked out of the basin."

Case 7.—Maryanne Neale, aet. 23, exposed by her mode of living to syphilitic infection, went to bed on Wednesday morning, June 2, 1845, quite well at 2 o'clock a. m., but awoke at 7 o'clock with great pain in the head, especially over the left frontal eminence. She then perceived that she had lost the power of her right arm and leg. She was freely bled for this and afterwards blistered, and on the 6th of June was admitted into the hospital in the following condition:—complete hemiplegia of the right side, involving the muscles of the face—the arm more palsied than the leg—speech indistinct—consciousness natural—sensation unimpaired.

There could be no doubt of lesion of the left side of the brain in this case, having probably a similar origin to that in the last case.

Here again the contractions produced by the galvanic current were distinctly much less powerful in the palsied limbs than in the sound limbs.

Case 8.—George Brosby, aet. 58. This patient had complete hemiplegia of the right side, which began with symptoms of softening, at which time the palsy was imperfect; this state lasted three weeks, when he became distinctly apoplectic with stertorous breathing; he recovered from this attack with complete paralysis of both right arm and leg. The following note was made of the only trial which was made with the galvanic current:—"The galvanic current, when passed through the sound leg, caused distinct action of the muscles, and the tibialis anticus was powerfully raised; but when applied to the paralysed limb, the action of the muscles was not nearly so strong. When a pole was applied to each ham, action was induced in both limbs, that of the

sound limb being decidedly the stronger. If the current was passed along the front of the thigh, the action of the anterior muscles was induced strongly on the sound side—*very feebly on the paralysed side.*

Case 9.—John Drummond, aet. 55, a tall muscular man, has hemiplegia of the left side, affecting the face as well as the extremities. Three years and a-half ago, he suddenly became hemiplegic without any impairment of consciousness; he had but just imperfectly recovered from this palsy when he was again attacked on awaking from sleep with palsy of the same parts. The radial arteries are thick, and several spots of deposit may be felt in them. It is probable that extensive disease of the arterial system exists, and that disease of this kind in the cerebral arteries has induced a white softening of a portion of the right hemisphere of the brain.

The galvanic current in this case affected both arm and leg, much less on the palsied side than on the sound one. Its influence on the arm was less even than that on the leg.

Case 10.—Mrs. L., aet. 53. This case occurred to me in private practice, and I am indebted for the opportunity of witnessing it to my friend Mr. Dunn, who is well acquainted with the present state of inquiry into the physiology and pathology of the nervous system, and takes a great interest in their progress.

The patient is a spare woman; she was found on Monday morning, the 17th May 1847, lying on the floor in her bedroom, speechless but without stertorous breathing, and not wholly unconscious—her consciousness gradually returned. I saw her four days afterwards, and found her completely hemiplegic, as to motion, on the right side—sensation unimpaired. Active reflex movements can be excited on tickling the foot, but none in the upper extremity. She yawns a great deal. There is no muscular rigidity whatever. Urine and faeces passed involuntarily. Puts her hand to the left side of the head.

The hands and afterwards the feet were placed in basins of water. I first used a Cruikshank's battery of fifty plates, but without any effect on either limb. I then had recourse to

the magneto-electric rotation machine: by this, active movements were excited in the healthy leg and arm; none in the palsied leg, very slight and feeble contractions in the palsied arm. On applying the poles directly to the palsied limbs contractions were excited, but even these were extremely feeble and partial.

This experiment was witnessed by Mr. Dunn.

On the 26th of May I repeated the experiment. The patient had acquired rather more consciousness and strength, but the palsied limbs were apparently unchanged. When the current was passed through both arms, by means of the basins, *only one or two muscles of the paralysed arm* were feebly excited, whereas the sound one was thrown into energetic action. The same results precisely were observed in the legs.

Mr. Dunn was present and assisted at this experiment likewise.

I am indebted for the next and the two following cases to my friend Dr. Babington, of Guy's Hospital, who requested Dr. Novelli, a very intelligent and competent manipulator, to apply the galvanism. Dr. Novelli has kindly furnished me with his notes of the experiments.

Case 11.—Eliz. Beaumont, *at. 33*, admitted for hemiplegia of six weeks duration. The whole of the right side was paralysed. Three weeks after her admission, *i. e.* nine weeks after the paralytic attack, the following experiment was made:—The hands were immersed each in a separate basin of warm water, which were in turn respectively connected with the electrodes of a magneto-electric machine of feeble power. *No effect whatever was produced upon the right arm*, but the muscles of the left, *i. e.* the sound limb, were attacked with spasmodic quiverings and involuntary contractions. The hands were now removed and the feet substituted. A precisely similar result was obtained; the muscles of the sound leg being excited to irregular and involuntary action, *while the paralysed limb remained quiescent*.

The patient left the hospital about a fortnight afterwards unbenefted by the treatment employed. Previous to her de-

parture I repeated the experiment. The phenomena were unchanged.

The hemiplegia had been preceded by pain in the head and loss of consciousness.

Case 12.—Charles Hutchins, æt. 33, came under Dr. Babbington's care on the 27th of May 1846. Three days before coming to the hospital, he had been seized with sudden pain on the left side of his head, and loss of consciousness, lasting for two hours and a half. Upon recovering from the fit, the left side of the body was found to be paralysed. I subjected this patient to the influence of voltaic electricity as above described, and it was again found that the sound arm exhibited feeble muscular contractions, *while the paralysed limb remained unaffected.*

At a *post-mortem* examination which took place, an abscess was found in the right hemisphere of the brain, involving the membranes, and extending within a few lines of the roof of the ventricle. Here, therefore, the paralysis had indubitably a cerebral origin.

Case 13.—Thomas Gardner, æt. 25, paralysis of the right arm of four months standing. In this instance, also, the paralysis was preceded by pain in the head and a fit. The same experiment was performed as in the two previous cases, and the same results were obtained.

I have now adduced thirteen cases which unequivocally demonstrate that, in certain morbid states of the brain, the contractility or irritability of the muscles of the paralysed limbs is not augmented. Setting aside all comparison between the muscles of the sound and palsied limb, there can be no doubt that if the irritability of the latter were augmented, the galvanic current ought to excite them to brisk contraction.* Yet the experiments showed that in some no effect whatever was produced, and in others the effect was only slight. It may be considered, therefore, as proved that the cutting off

* In experiments with the galvanic current upon muscles, the contractions are always in the direct ratio of the intensity of the current.

of the brain's influence from a muscle does not (in every case at least) lead to the augmentation of its irritability.

There are, however, cases of hemiplegic and even of paraplegic paralysis in which the muscles respond very readily and vigorously to the galvanic stimulus, and even display a greater amount of vigour than the muscles of the healthy limbs. In these cases the muscles of the palsied limb always exhibit some degree of rigidity, and the vigour of their action in obedience to the galvanic stimulus will be proportionate to the amount of rigidity, within certain limits. It must be borne in mind, however, that this is the case generally only where the rigidity is recent, and appeared along with or very shortly after the paralytic seizure.

It is important to make a distinction between the rigidity which affects the paralytic limb early, and that which follows the paralytic seizure at a distant interval. The latter is indicative of loss of substance in the nervous centre. A patient has softening or a clot; this produces paralysis; by and bye the softened brain shrinks, or the clot becomes absorbed, and the surrounding healthy brain gradually contracts around the lesion, and as it contracts it causes rigidity of the paralysed muscles. The early rigidity of the palsied muscles accompanies a state of irritation of the brain, and will disappear when that irritation is subdued. Red softening, a tumour, meningeal disease, inflammation around a clot, are all capable of producing this state of muscle. This early rigidity may be taken as indicative of an augmented innervation of the affected muscle, which is thereby kept in an excited state.

Again, there are other cases in which, while the paralysis is pretty complete, the galvanic stimulus excites equally the muscles of the sound and those of the healthy limbs. These are generally cases of apoplexy occurring in persons previously healthy and not advanced in years. The muscles are healthy and well nourished; and, after the first shock has subsided, they respond readily to the galvanic stimulus, but not more so than those of the sound limb.

I shall quote some cases in illustration of these remarks.

Case 14.—Patrick Cochrane, æt. 52. This man had enjoyed but indifferent health for some months before his admission, having suffered from irregular gout. On the 10th of November, at five in the afternoon, he suddenly experienced a creeping sensation, commencing in his head and extending to the left half of his body; he became giddy and sick, and, on attempting to rise, found that he had lost the use of his left arm and leg; the left side of the face was paralysed and the tongue protruded to the left side, and he had great difficulty of deglutition. Intellect slightly impaired.

There was slight rigidity of the muscles of both arm and leg, and these limbs also started a great deal both by day and night—a symptom most troublesome to the patient. These startings were brought on by yawning, and, on such occasions, were most conspicuous in the upper extremity.

After he had been in the hospital for ten days, during the last four or five of which the startings of the limbs were very great, the following trials were made with the galvanic apparatus.

The galvanic current was first passed from the popliteal space to the dorsum of the left (or paralysed) foot, and through the muscles of the left thigh and left calf. It was then passed, as nearly as possible, between the same points on the right or healthy side. The muscular contractions produced were most active and extensive *on the left or paralysed side.*

The current was then passed along the median nerves of the upper extremities and through their muscles. The contractions produced were clearly more powerful on the left or paralysed side than on the right.

The rigidity and startings became less in the course of a few days, and on the 7th of December it was reported that the arm was relaxed and soft, but the leg rigid so as to prevent complete extension. The galvanism excited now greater contraction in the paralysed leg than in the sound one, but less in the paralysed than in the sound arm.

The startings in the paralysed limbs ceased altogether on the 28th; he was regaining power in the leg, but the arm

had wasted, and hung quite flaccid. On the 14th of January I gave him some strychnine, in doses of one-sixteenth of a grain, three times a-day. Four days afterwards he began to feel a pricking sensation in the left foot, leg, and thigh, and the left side of his hip, followed by startings of the leg ; there was no sensation referred to the arm. It was not until twelve days after this that the arm began to be affected by cramps, although he had experienced uneasy sensations at an earlier date.

Case 15.—Robert Morgan, *æt. 25*, a clerk. Three weeks before his admission he had violent pains in the head, preventing him from sleeping, accompanied with thirst and fever. These subsided and recurred, and again subsided and recurred : he was then attacked with vomiting ; his speech became affected, and on the 13th of November 1845 his right arm and leg became powerless, and consciousness was much impaired.

The paralysis was complete in both extremities, but the muscles of the upper extremity presented a considerable degree of rigidity. The right fore-arm was firmly bent on the arm by the action of the biceps, and the fingers and thumb were flexed into the palm.

I had no doubt, in this case, of the existence of red softening in the left hemisphere of the brain.

On applying the galvanic current, marked contractions of the right (or paralysed) arm were produced. Very little effect was produced on the right (paralysed) leg, but there was considerable action of the left leg.

Case 16.—Eliza George, *æt. 44*. In this case there was hemiplegia of the right side. On awakening in the morning she was seized with dizziness, and her right leg became paralysed : three days afterwards the arm became affected. The intellect was much impaired and articulation destroyed, so that she could only utter the monosyllable "No." There was rigidity of the muscles of both limbs, especially of the upper extremity.

The effect of the application of the galvanism is thus noted : "the affected arm was more convulsed and more painful than

the sound one. The same effect was produced in the leg, but to a less degree."

In addition to these cases, I may state that, about three years ago (July 5, 1844), Dr. M. Hall did me the favour to send me a patient, in whom he stated that galvanism excited more contraction in the paralysed than in the sound limb. In this patient, a girl, æt. 15, there was paralysis of the right arm, with *contraction* and *rigidity* of the flexor muscles and inability to extend the fingers.

Dr. Novelli has furnished me with an account of his trial of galvanism on a case of recent paralysis of the left arm. In this case he used a trough of fifty plates. Small plates of zinc were applied at certain points of the limbs, and to one of these each wire of the battery was applied, contact being made and rapidly broken by touching one of the zinc plates with one of the wires, and immediately removing it. The result was, that no difference whatever was observable as to the degree of contraction produced in either limb—although the experiment was performed with great care, and repeated several times with the like result.

I have tested two cases of recent hemiplegia after apoplexy by the galvanic current, with the result of finding very slight difference in the contractions of the two limbs; the palsied limbs, however, exhibiting feebler contractile power than the sound ones.

Nyrten relates that he tested the muscles with galvanism in two apoplectics, who died some days after the attack; and that there was as much contraction in the paralysed as in the sound limbs.*

* I had made several experiments on this subject, and had frequently expressed my doubts of the correctness of Dr. Marshall Hall's views, before I was aware that my learned friend, Dr. Pereira, had formed similar opinions. His valuable work on *Materia Medica* is so generally in the hands of medical readers, that I need not quote the remarks which he makes on this subject. I shall only add, that he relates one case of cerebral palsy in which the paralysed muscles were less excitable to the galvanic stimulus than the healthy ones; and he states that he has "observed the same effects in many other cases."—Pereira, *Mat. Med.*, vol. ii. p. 1301.

I have thus referred to three classes of cases, in each of which the paralytic limbs respond differently to the galvanic stimulus. In the first class the stimulus produces little or no contraction; in the second it causes vigorous contractions, and even of a more lively character than those in the sound limbs; and in the third, contractions are excited of a more or less vigorous kind, but which exhibit little or no difference from those of the healthy limb.

In the first class of cases, the paralysed muscles may be more or less wasted, or they may present no difference in point of nutrition from those of the sound limb. In my tenth case there was no difference, as regards the nutrition of the muscles, between the sound and palsied limbs; yet the electric current excited scarcely any contractions in the latter.

In the second class, the paralysed muscles at first exhibit no loss of nutrition—on the contrary, they are manifestly firmer than the healthy muscles; if the palsy yields to treatment, they lose this increased firmness or rigidity and assume the natural condition; if, on the other hand, the disease obtains the mastery, the muscles waste, although in some instances they continue to maintain their rigidity.

In the third class, the muscles retain their normal condition, and will continue to do so if the patient is not slow in recovery.

In reviewing the results of the experiments in these cases, this conclusion forces itself upon me—that the effect of the galvanic stimulus upon a paralytic limb is always feeble when the nutrient condition of the muscles is low, i. e. when there has been much wasting of the muscles; but that a well-nourished state of the muscles in a paralysed limb is no index of the probable amount of contraction which the galvanic stimulus would excite in them.

A little reflection upon this conclusion suggests that, in all probability, the state of the muscles has essentially little to do with the phenomena excited by the galvanic stimulus; else, in every instance the state of the muscle and the effect of the galvanism ought to correspond.

To explain the phenomena we must look to the state of the *nerves* of the paralytic limb. An analysis of the cases shows that a direct relation exists between the degree of development of the *nervous force* in the nerves of the affected limb and the effect of the galvanic stimulus.

In the first class of cases the nervous force is depressed in the nerves of the paralytic limb ; in the second class it is exalted ; and in the third class it retains its normal development.

Every one who has experience in physiological experiments upon the nerves and muscles, knows that the application of the galvanic stimulus directly to the muscle itself is much less effective in exciting it to contract, than the application of it to the nerve by which the muscle is supplied. When we observe how general and consentaneous is the action of the muscles in the upper or lower extremities, when the galvanic current is passed through them, each limb being immersed in a basin of water according to the method described in the experiments, it is impossible not to conclude that it is upon the nerves that the galvanic stimulus acts under these circumstances, and that the contractions of the muscles are the result of its influence upon the nerves. In corroboration of this view, let the poles of the electric apparatus be applied to the muscles themselves, and the contractions excited are comparatively feeble ; but let them be applied in the course of some of the principal nerves of the limb, as the median or the sciatic, and a marked increase in the number and vigour of the contractions will be produced.

This view acquires further support from the following experiment. Let the posterior extremities of a frog be separated from the trunk at the dorsal region of the spine, and then let them be skinned. If the feet be immersed each in a cup or glass of water, and a weak current passed through the limbs, both will be equally affected : let now a piece be cut out of the sciatic nerve in one thigh,* and immediately the contrac-

* Very conclusive evidence in favour of this view is derived from Matteucci's experiments, which show that, after a time, *the direction* of the current exerts an important influence upon the contractions. This in-

tions in that limb will be manifestly less than those in the uninjured limb.

We must look then to the state of the nerve to explain the different effect of the electric current upon the muscles. When the nerve is in a state of irritation, or, in other words, when the nervous force in it is in a *plus* condition, so to speak, the galvanic stimulus will take great effect ; on the other hand, when the nervous force is depressed, or in a *minus* condition, the galvanic current takes little or no effect.

Galvanism may, therefore, sometimes serve as a test to distinguish between an irritant and a depressing lesion of the brain. In using this test, however, the practitioner will do well to bear in mind, that an irritant disease of the brain is not necessarily inflammatory, but that, on the contrary, inflammation of the brain is very frequently most depressing in its effects. A spiculum of bone or a meningeal tumour may be irritant in its effects, but not inflammatory ; whilst a patch of red softening, which is distinctly inflammatory, will often produce great depression, both local and general.

These views suggest what appears to me to be the true explanation of the remarkable tendency of strychnine to affect paralytic limbs first. It will scarcely be doubted, now-a-days, that a medicine which exercises such potent influence upon the body, when given in so minute doses as one-eighth or one-fourth of a grain, must do so through the blood. This material, being taken up into the blood, circulates with that fluid wherever it flows, to all parts of the cerebro-spinal centre, equally on both sides, if the circulation on both sides be in equilibrium. But if there be any lesion on one side of the brain, which from the morbid irritation, or for purposes of repair, attracts a larger quantity of blood to or around it than that which flows to the corresponding part on the opposite side, then a greater amount of the strychnine will be accumulated there, and that part will become the seat of irritation

fluence is clearly exerted through the nerve, for in applying galvanism to a muscle, it is of no consequence in what direction the current may pass.

by that drug. This irritation will, in conformity with the known law of cerebral action, be communicated to the muscles of the opposite or paralysed side, and consequently the muscles of that side will first exhibit the peculiar effects of strychnine.*

In a few rare cases, strychnine does not affect the paralysed limbs first. In these cases the lesion in the brain is doubtless of such a nature that the blood is attracted to the brain-substance around it in less quantity, than to the corresponding parts of the opposite side.

What, then, is the condition of the irritability or contractility of the muscles of the paralysed as contrasted with those of the sound limb? Certainly it is not augmented. It is in exact proportion to the nutrition of the muscles. Matteucci's extensive and carefully conducted experiments have quite settled the much-vexed question respecting the *vis insita*. He has shown that the nutritive changes of muscle develope a galvanic current, which passes from the interior to the surface of the muscle, and that muscular contraction causes what may, with the highest probability, be regarded

* There are many pathological facts which show that a poison in the blood is apt to be attracted largely to any part to which there may be a great flow of blood from any previous cause. Four years ago I attended a lady in small-pox; she had in one groin a large congenital nævoid patch, and was much alarmed to find that this spot had an extraordinary accumulation of pustules upon it. A very similar case is related in Dr. Wm. Budd's paper in the 25th volume of the Society's Transactions, p. 129. Ollivier justly remarks, that it is not the motor power only which is affected by strychnine, but also sensibility; that paralytic limbs are frequently rendered painful by strychnine, and even before the motor power becomes affected. In the fourteenth case referred to in this paper, this effect of strychnine is recorded; and I believe this to be the most common way in which this drug begins to show its effects. Many physicians write and speak of this poison as if it had a peculiar and specific influence on the spinal cord, *to the exclusion* of other parts of the cerebro-spinal centres. This view I believe to be incorrect, and that the influence of strychnine pervades the whole cerebro-spinal system; it by no means follows that, because this drug *appears* to act upon the spinal cord first, it acts *exclusively* upon that organ.

as an electric discharge, which can excite the nervous force in the nerve of a frog's leg,—and that these phenomena are in the direct ratio of the nutritive actions of the muscle,—they fail when the changes of nutrition are slow and imperfect or vitiated, and they are distinct and easily demonstrable when the muscles are well nourished. And when we take into account the beautiful and complicated structure of muscle, which modern research has so well developed,—when, likewise, we remember its complex and peculiar chemical constitution, it is impossible to refer the force which muscles can develope to any other source than its own tissue and the chemical and physical changes of which it is the seat. The two tissues, muscle and nerve, and the forces which they respectively develope, are, no doubt, nearly allied, and probably so far mutually dependent, that the imperfect nutrition of either would exert an unfavourable influence upon the other.

It is in vain to found any distinction between cerebral and spinal palsy, upon any difference that may exist between the paralysed and healthy muscles as to their excitability by galvanism. The very same states of muscle and of nerve exist in spinal palsies as in cerebral. There is this difference, however; that as in spinal palsy the nerves are more nearly related to the seat of lesion, sometimes, indeed, implanted in it, they participate more directly and, therefore, more completely in the effects of the lesion than in cerebral palsy; and when these effects are depressing, the nervous force is more completely depressed, or when they are irritant, the nervous force is more exalted, in spinal than in cerebral palsies. In a case of intra-spinal disease, which occurred about twelve months ago in King's College Hospital, the lower extremities were in a state of continued tonic contraction more intense than I had ever seen them in tetanus. These muscles responded readily and very forcibly to the galvanic stimulus. Yet the influence of the will was completely withdrawn from them, and the paralysis of voluntary motion and of sensation was complete. Muscular rigidity to a less degree is by no means uncommon in spinal palsy.

Furthermore, it may be stated that strychnine is as apt to affect the paralytic limbs where the lesion is spinal as where it is cerebral.

In conclusion, I will sum up the results of the preceding observations in the following propositions :—

1. The contractility or irritability of the muscles of paralysed limbs bears a direct relation to their state of nutrition.

2. The excitability of the paralysed muscles to galvanism varies with the condition of their nerves, more than with that of the muscles themselves.

3. In the majority of cases of cerebral palsy, the contractility or irritability of the paralysed muscles is less than those of the sound side, simply because their nutrition is impaired by want of exercise.

4. No diagnostic mark, to distinguish between cerebral and spinal palsy, can be based on any difference in the irritability of the paralysed muscles ; for the muscles in spinal paralysis exhibit the same states as those in cerebral paralysis.

5. The tendency of strychnine to affect the paralytic limbs before the healthy ones, is attributable to its being attracted in greater quantity to the seat of lesion in the brain than to the corresponding part on the other side.

6. The manner in which the muscles of a paralysed limb respond to the galvanic stimulus, assists us in forming an opinion as to the state of its nerves ; if they respond feebly or not at all, the nerves are in a depressed state : if they respond vigorously, more so than the healthy muscle, the nerves are in a state of irritation.

FATAL CASE
OF
DYSPHAGIA;
PRODUCED BY A POLYPOUS GROWTH IN THE
ŒSOPHAGUS.

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COMMUNICATED BY RICHARD PARTRIDGE, F.R.S.,
VICE-PRESIDBNT OF THE SOCIETY.

Received June 20th—Read June 22nd, 1847.

JAMES KELSEY, aet. 48, a ribbon-weaver, tall, spare, not intemperate nor previously unhealthy, applied to Dr. Phillips of this city, at the end of the first week in January last, on account of sore throat and difficulty of swallowing. On the 3rd of January, in consequence, as he supposed, of food disagreeing with him, he had vomited violently, and he observed to his family at the time, that he thought he had injured his throat by it. On inspection of the throat, no signs of disease could be discovered. He received some simple treatment, and returned to his employment in the course of about a fortnight. He continued at work for the space of three weeks, the complaint gradually returning and increasing, till at length the dysphagia became severe.

About the beginning of March he was entered as an out-patient at the Coventry Hospital, under my care. He had then much difficulty in swallowing, with frequent and, at times, severe cough (always excited by attempts to swallow,

as well as occurring independently of them), very copious frothy expectoration, fever, but no dyspnoea. The tonsils, uvula and pharynx were healthy. In examining the throat every endeavour was used to bring the epiglottis into view, but in vain, the narrowness of the man's mouth contributing to the difficulty of the examination. The stroke-sound of the chest was everywhere moderately clear, the respiratory murmur uniformly feeble, but without *râle*. On applying the stethoscope over the front of the neck, the inspiratory sound was heard preternaturally loud and rough. At this time it was conjectured that some result or product of inflammation occupied the commencement of the œsophagus, and permitted the glottis to remain unclosed during deglutition.

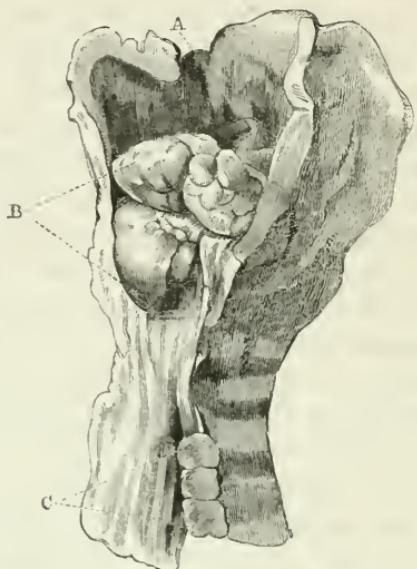
In a few weeks the feverishness and cough (excepting when attempts were made to swallow) had very much subsided, but the dysphagia had progressively increased, till, at length, deglutition became nearly impossible, and the man was evidently sinking from inanition. The only position indeed in which he could at all swallow was, by lying decumbent and then throwing his head farther back. In this way some portion of fluid could be got down. Attempts were several times made to pass a tube into the œsophagus, but these uniformly failed. They were followed by some bloody excretion, and occasioned, at the moment, so much spasm of the glottis, as apparently to endanger suffocation.

As the efforts to swallow and to pass instruments into the œsophagus appeared to be mainly frustrated by spasm of the glottis, and impediments to inspiration thereby induced, it occurred to me as not improbable that, if tracheotomy were performed, respiration might be carried on through a tube left in the trachea, with far less impediment, whilst more deliberate and persevering attempts to swallow and to pass instruments were made. Accordingly, on the 14th of April, the operation was performed, and with much skill, by Dr. Dewes, the resident medical officer of the hospital; the trachea tube was inserted and secured, and allowed to remain to the period of the patient's death.

Although the attempts to pass a tube into the œsophagus proved equally unavailing after the operation as before it, and were but little less irritating, the power of deglutition a good deal improved in two or three days, and for nearly a week afterwards he was able to swallow half a pint of milk at a time. Yet, from the period of the operation, whenever he swallowed, even most successfully, some portion of the fluid always returned through the tube in the trachea, showing that the glottis was imperfectly closed. After the lapse of about a week the dysphagia again increased, and swallowing soon became quite impracticable, and death ensued purely from inanition. For eight or nine days preceding his death he never swallowed anything, yet he vomited bitter yellow fluid occasionally, which passed upwards without unusual difficulty. Previous to the performance of tracheotomy and subsequently, nutritive enemata were regularly administered. The pangs of hunger were the chief source of suffering, and even these and an occasional cough were mitigated by sucking syrup of morphia. Death took place on the 14th of May, about fourteen weeks from the apparent commencement of the disease.

The *post-mortem* examination was made twenty-two hours after death by Dr. Dewes, assisted by Dr. Phillips. The tongue, larynx, trachea and pharynx were removed in connection, and the cause of death became at once revealed. A polypous growth was discovered at the commencement of the œsophagus immediately behind the glottis. The tumour was lobulated, freely mobile, of a sub-livid colour and cellular texture, and about two inches in length at the periphery. In bulk it was somewhat larger than a walnut, and was attached to the mucous tissue by a short thinish fibrous base, commencing about half an inch from the posterior commissure of the glottis, and extending for the same distance in a straight line in the axis of the œsophagus. This fibrous attachment extended laterally also for about three quarters of an inch towards the right, in a direction between the hyoid bone

and thyroid cartilage. The structure of the tumour was vascular and homogeneous.



A. The glottis. B. The larger polypoid growth. C. The smaller tumour.

This tumour formed on the one hand a mechanical obstacle to the perviousness of the oesophagus, and on the other, by passing under the epiglottis during attempts to swallow, prevented the closure of the glottis, and thus allowed fluids to pass into the trachea. Another smaller tumour of very white colour, about three quarters of an inch long, and attached by a broad base, was found nearly two inches lower down the oesophagus. The epiglottis, larynx and trachea were free from disease. The pleurae and lungs were everywhere sound, with the exception of a limited tubercular deposit, of granitic aspect, in the apex of the right lung. The further examination of the body was prohibited by the friends, some of whom were present during the inspection, but the morbid specimen was brought away.

The case now related differs from any other instance of

polypus of the œsophagus which I have seen referred to, in the dysphagia being complicated with, and greatly increased by, the tendency of food to pass into the glottis, and thereby to induce distressing cough and to threaten asphyxia, a circumstance simply due to the peculiar position and form of the morbid growth. Polypi in this situation must, therefore, be exceedingly rare; indeed polypi of the œsophagus are probably of less frequent occurrence than in any other of the open cavities of the body. (Portal—*Memoires sur plusieurs Maladies*, 1808, tom. iii.) Yet they are distinctly referred to by writers on Morbid Anatomy, as by Otto (*Handbuch des Path. Anatomic*. Breslau, 1814, s. 257) and Monro (*Morbid Anatomy of the Gullet, &c.*). An interesting case from this latter work is copied by Hodgkin (*Lectures on the Morbid Anatomy of the Serous and Mucous Membranes*, ii. 249), and, indeed, is referred to by almost every other author on the subject since it appeared. Blandin quotes a case from Schneider of a woman who died from dysphagia, occasioned by two polypi in the œsophagus, the existence of which had not been suspected during life. This author adds, with reference to the disease, “*leur diagnostic est d'une désespérante obscurité.*” (*Dict. de Medic. et de Chir. Pratiq.* xiii.) It seems right, therefore, that the present case should be recorded as a contribution towards the removal of that obscurity in diagnosis which Blandin, in another place, pronounces to be inevitable, but which careful and extended observation may hereafter elucidate.

N O T E.

The following note has been prepared by direction of the Council. The facts and statements it contains have reference to the subject of the papers of Mr. Simon and Dr. Johnson, on the inflammatory diseases of the kidney.—ED.

1. The existence of an inflammatory affection of the kidney, included under the name of *Morbus Brightii*, but distinct from the cirrhosis or fatty disease of the kidney, was admitted, on microscopical grounds, by Glüge, in his *Anatomisch-Mikroskop. Untersuchungen*, Heft ii. Jena, 1841.

It is clearly described by Vogel, in his “*Icones Histologicae Pathologicæ*,” 1843, page 108. In his explanation of table xxiii. he says: “Fig. 4. illustrates an inflammatory disease of the kidneys, which is not rare, and which can be recognized generally only by microscopical examination of the urine. The urine,—sometimes bloody, sometimes of normal colours, but always turbid in the early stage,—deposits a whitish yellow sediment. . . . The urine sometimes contains much albumen, and then coagulates on boiling: sometimes the albumen is in small proportion, but it rarely is absent. The sediment, when examined by the microscope, is seen to be formed of colourless coagula of cylindrical form, the diameter and shape of which correspond exactly to the secreting tubuli of the kidneys. They contain, like those tubuli, particles of epithelium, and sometimes, also, rust-coloured granules (altered blood). Those coagula are easily soluble in caustic potash, and, less readily, in acetic acid;

but the latter re-agent renders the epithelial cells contained in them more distinct. It can scarcely be doubted that these cylindrical bodies are coagula of fibrin, which form in the renal tubuli, including a part of the epithelium of those canals within them, and which preserve the form of the tubuli when discharged from them. They have no constant length: often they are several lines long; their thickness varies from $\frac{1}{200}$ to $\frac{1}{90}$ of a line. Sometimes pus globules in large number are mingled with these cylinders. . . . I have seen these appearances in ten patients. Symptoms of much disease, especially pain on deep pressure in the lumbar region, may always be detected by careful examination."

2. In describing the urine in *Morbus Brightii*, Franz Simon (Simon's *Beiträge*, p. 103) describes the sediment as consisting of "mucous corpuscles," "epithelium from the bladder," "blood-discs," "dark globules, apparently filled with a granular substance," and the cylindrical bodies which, he says, are "composed of a finely granular substance including cells and globules similar to mucous globules," and which he regards as the altered epithelial lining of the secreting tubuli of the kidneys. He urges the importance of these cylinders as signs of *Morbus Brightii*. In a case supposed to be one of *scarlatina*, he found in the urine blood-discs, a great number of the cylinders, mucous globules and crystals, and globules of lithic acid. The cylinders, too, he says, contained crystalline matter resembling lithic acid. He could not from the urine decide whether this was a case of *scarlatina*, but he could say, with confidence, that there was great irritation of the kidneys which might have been produced by too abundant deposit of lithic acid.—(P. 110.)

Heller (Archiv. für Physiol. u. Pathol. Chemie und Mikroskopie, Band ii. p. 173) gives a very similar account of the constant components of the urinary sediment in Bright's disease, or dropsy with albuminous urine, and shows that the epithelium of the tubuli uriniferi is thrown off in a tubular form.

Scherer (Chemische und Mikroskopische Untersuchungen.

Heidelberg, 1843) describes the same characters of the urine in Bright's disease, in a case of haematuria, followed by albuminuria, and also in the desquamative stage of scarlatina. He describes the cylindrical bodies in the urine as resembling false membranes, like the membranes in croup, thrown off from the minute renal tubuli.

3. The tubuli of the kidney, after death from Bright's disease, were found filled with amorphous matter, coagulated albumen, or fibrin, by Valentin, and afterwards by Henle.

Valentin (*Repertorium*, 1837, p. 290) describes the tubuli as being filled with a yellowish grey mass, which was composed of irregularly granulated portions of different sizes, small molecular corpuscles, and round yellowish globules; the walls and interstices of the canals being healthy.

In the *Repertorium*, 1838, p. 185, he says, the disease consists in exudation of albumen in abnormal quantity; that a part of it remains dissolved and is discharged, while a part is precipitated, and fills the tubuli.

Henle (*Henle und Pfeuffer's Zeitschrift*, Bd. i. 1842, p. 60) found the tubuli of the kidney in part filled with matter, which he regarded as coagulated fibrin; though this matter was exuded in greater quantity in the interstices of the tubuli. In the 2nd vol. of the same Journal (p. 272), Henle, in noticing Glüge's statement that the blood-vessels of the cortical structure of the kidney, in the inflammatory form of Bright's disease, were filled with inflammation corpuscles, says, "what Glüge takes for blood-vessels filled in parts with morbid products, are urinary tubuli containing here and there blood."

Concerning the partial obliteration of the tubular structure of the kidney, and infarction of the tubuli with albuminous matter in scarlatina, jaundice, &c., Mr. Busk's paper, in the last volume of the *Transactions*, may be advantageously consulted.

DONATIONS

TO THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY,

1846-7.

* * * Those works which have been presented by their respective Authors are distinguished by an Asterisk.

ACTON, WILLIAM.

Essay on the great prevalence of Venereal Diseases in Great Britain, by Alfred Hall, M.D. 8vo. Glasgow, 1847.

ALDERSON, JAMES, M.D., F.R.S.

*Practical Observations on some of the Diseases of the Stomach and Alimentary Canal. 8vo. London, 1847.

ALDIS, C. J. B., M.D.

Health of Towns Association. Report of the Committee to the Members of the Association, on Lord Lincoln's Sewerage, Drainage, &c., of Towns Bill. 8vo. London, 1846.

Health of Towns Association. Unhealthiness of Towns, its Causes and Remedies ; being a Lecture delivered on the 10th of December 1845, in the Mechanics' Institute at Plymouth, by Viscount Ebrington, M.P. 12mo. London, 1846.

Letters on the unhealthy condition of the lower class of dwellings, especially in large Towns, by the Rev. Charles Girdlestone, A.M. 8vo. London, 1845.

BALY, WILLIAM, M.D., F.R.S.

*On the Pathology and Treatment of Dysentery, being the Gulstonian Lectures delivered at the College of Physicians in February 1847. 8vo. London, 1847.

BARKER, J. HERBERT, M.B.

*A Case of Large Secondary Prostatic Calculus, removed by Perineal Incision. 8vo. Worcester, 1847.

BARROW, BENJAMIN.

*Description of an Apparatus for Injuries and Diseases of Joints; with Observations and Cases. 8vo. Worcester, 1847.

BECK, THOMAS SNOW.

*On the Nerves of the Uterus. 4to. London, 1846.

BUSHNAN, J. STEVENSON, M.D.

*Observations on Hydropathy, with an account of the principal Cold Water establishments of Germany. 8vo. London, 1846.

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*Elementi di Medicina Practica Scritti. 4 vols. 8vo. Napoli, 1841-1844.

CHILD, GEORGE C., M.D.

*On Indigestion and certain Biliary Disorders often conjoined with it; to which are added, short notes on Diet. 8vo. London, 1847.

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COOPER, WM. WHITE.

*Practical Remarks on near Sight, aged Sight, and impaired Vision; with Observations upon the use of Glasses and on Artificial light. 8vo. London, 1847.

CORMACK, JOHN ROSE, M.D.

*Natural History, Pathology, and Treatment of the Epidemic Fever at present prevailing in Edinburgh and other towns. Illustrated by Cases and Dissections. 8vo. London, 1843.

*Contributions to Pathology, Therapeutics, and Forensic Medicine. 8vo. London, 1844.

*Correspondence between the Managers of the Royal Infirmary, Edinburgh, and Dr. Cormack, relative to the resignation of the latter as one of the Physicians of the Institution. 8vo. Edinburgh, 1845.

*Remarks on Dr. Handyside's Case of Suicide. 8vo. Edinburgh, 1838.

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*A Memoir on Amputation of the Thigh at the Hip-joint (with a successful Case). Folio. London, 1845.

*Operative Surgery, Mainganlt's. Illustrations of the Different Amputations performed on the Human Body, represented by Plates designed after Nature, with Alterations and Practical Observations. Second Edition. Folio. London, 1845.

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Hunterian Orations. 4to. London, 1814.

A Faithful Record of the Miraculous Case of Mary Jobson, by W. Reid Clanny, M.D., F.R.S. 2nd Edition, 8vo. Newcastle-upon-Tyne, 1841.

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A Descriptive Catalogue of the Anatomical Museum of St. Bartholomew's Hospital, vol. i., containing the Descriptions of the Specimens illustrative of Pathological Anatomy. 8vo. London, 1846.

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*Remarks on the Diet of Children, and on the Distinctions between the Digestive Powers of the Infant and the Adult. 12mo. London, 1847.

GREEN, JOSEPH HENRY, F.R.S.

*Mental Dynamics, or Groundwork of a Professional Education; the Hunterian Oration, before the Royal College of Surgeons of England, 15th February 1847. 8vo. London, 1847.

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*On Medical Education: being a Lecture delivered at King's College, London, at the opening of the Medical Session 1846-7; to which is added, a Lecture delivered on the same occasion in the year 1844. 8vo. London, 1846.

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*Practical Observations and Suggestions in Medicine. Second Series, 8vo. London, 1846.

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Thirteenth Annual Report of the Trustees of the State Lunatic Hospital at Worcester, U. S., December 1845. 8vo. Boston, 1846.

The Twenty-second Annual Report of the Officers of the Retreat for the Insane at Hartford, Con. May, 1846. 8vo. Hartford, 1846.

Minutes of the Proceedings of the National Medical Convention, held in the city of New York, in the month of May 1846. 8vo. New York, 1846.

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*Theorie des Névroviscérites ou Fièvres Primitives.
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LEE, THOMAS S.

*On Tumours of the Uterus and its Appendages. (Jacksonian Prize Dissertation.) 8vo. London, 1847.

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